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NBDE PART II

A STUDENT-TO-STUDENT GUIDE

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 - ▶ Exam preparation advice and test-taking strategies from veterans ◀
 - ▶ Case studies and NBDE-style questions ◀

JASON E. PORTNOF ■ TIMOTHY LEUNG
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FIRST AID FOR THE®

NBDE Part II

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Medical

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PREFACE

With *First Aid for the NBDE Part II*, we continue the *First Aid* commitment to providing students with the most useful and up-to-date exam preparation guides. This new addition to the *First Aid* series represents an outstanding effort by a talented group of authors and includes the following:

- Frequently tested information and figures based on student experience.
- High-yield images, diagrams, and tables.
- Key Facts and helpful mnemonics.
- A high-yield collection of glossy photos similar to those appearing on the *NBDE Part II* exam.
- Case Studies and NBDE-style questions.

This first edition would not have been possible without the help of the students and faculty members who contributed their feedback and suggestions. We invite you to share your thoughts and ideas with us via email at **firstaidnbde2@gmail.com**, to help us improve *First Aid for the NBDE Part II*. Good luck on your boards!

<i>New York</i>	Jason E. Portnof
<i>New York</i>	Timothy Leung

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► INTRODUCTION TO THE NBDE PART II

Passing the National Board Dental Examination (NBDE) Parts I and II is a basic requirement for initial dental licensure in the United States. Most dental students take the Part II exam during their final year of dental school.

The National Board Dental Examination Part II is a comprehensive examination that tests the material you have learned in both your preclinical and clinical years. The Part II examination comprises of 500 multiple choice test items in total. The basic structure of the NBDE Part II is divided into two sections. The discipline-based component (Component A) consists of 400 items, and the case-based component (Component B) includes 100 items based on approximately 10 case problems. 70% of these Component B cases are based on adults and 30% deal with pediatric issues. The test has been developed so that 15% of Component B test questions will cover topics related to the management of medically compromised adults and children.

The clinical dental sciences tested in the NBDE Part II include operative dentistry, pharmacology, endodontics, periodontics, oral and maxillofacial surgery and pain control, prosthodontics, orthodontics, pediatric dentistry and oral diagnosis, oral pathology/dental radiology, and patient management (behavioral science/dental public health and occupational safety). About 30% of the items tested in NBDE Part II will have references applicable to the basic sciences and 30% will be multidisciplinary.

The test is no longer offered in a print format. However, the computerized NBDE Part II is offered all year-round by Thompson Prometric at Prometric Testing Centers. The examination must be scheduled on 2 consecutive days. Information about NBDE Part II format, eligibility, registration, and scoring can be found at www.ada.org

► COMPUTER-BASED TESTING BASICS

Candidates planning to take the NBDE Part II must submit an application and fee to the Joint Commission on National Dental Examinations. Once the application is processed, eligible test takers can register for the NBDE Part II testing appointment through Thompson Prometric (<http://www.prometric.com>) or by calling the Thompson Prometric National Registration Center 800-phone number.

On the day of the exam, you will be required to bring two forms of identification. One form of identity (ID) will require both a photograph and a signature. Passports or drivers licenses are always acceptable forms of primary identification. The names on the IDs must match exactly with the name on the candidate's Part II application file. The test taker should also know their Social Security or Social Insurance number.

Candidates will be photographed and fingerprinted at the Prometric Testing Center. The test is administered under strict supervision and security.

Prior to beginning the actual examination, the candidate is given the opportunity to take a brief tutorial. This will help familiarize the test taker with the computerized format.

► DEFINING YOUR GOAL

The first step you must take as an NBDE test-taker is define your individual goal for the examination. You can tailor your study style and intensity depending on your performance goals. By determining how high you want to score

on the exam, you can establish how much study time and preparation you will require.

Dental students taking the NBDE Part II have varied goals and utilize various different study techniques to meet these different goals. Determine your personal goal and define what is driving you toward that goal.

Your goal may depend on the specialty to which you plan on applying. Some specialty programs will require very competitive Part II board scores. Approaching senior dental students who have already undergone the application process can help you determine individual specialty requirements. You may also want to improve on previous scores in an effort to improve your application profile.

► SCORING OF THE NBDE PART II

The score of your NBDE Part II exam is dependent upon two factors. Your raw score (the number of correct answers you selected) and the score scale conversion for the examination. A standard-setting committee reviews the content of the exam to determine test scoring. Standard scores range from 49 to 99. The minimum passing score for the exam is 75. Partial credit is not available.

Whatever your goals may be, it is important to consider your goals early on in your study period so that you can prepare for the NBDE Part II accordingly.

Some possible test taking goals include:

1. Purely passing—Some NBDE test takers are concerned with simply passing the test. For example, you may not be planning to go into a residency program, and passing the NBDE Part II is all that you need.
2. Acing the exam—Other NBDE examinees will strive to “ace” the exam. Perhaps you want to strive for the highest score on the exam as possible. This is a good strategy if you are planning on applying for competitive residency or postgraduate study programs.
3. Evaluating your clinical knowledge—Using the NBDE Part II as an assessment of your knowledge of clinical dental sciences is good review prior to residency and postgraduate study. If you are not planning on postgraduate study, the NBDE Part II is a good tool to examine your ability to recognize classic clinical presentations and provide viable treatment to your future patients. The NBDE Part II can be appreciated as the milestone that examines all that you have diligently learned in your years of dental school.

► STUDY MATERIALS

Practice Tests

Practice tests provide valuable information about strengths and weaknesses of your knowledge base. They also give you insight as to how questions will be asked, which can help focus the direction of your studies. Practice questions also help to break up the monotony of reading review material. Regardless of why people use practice tests, it is important to use practice tests and to begin them early in your study schedule. Old released editions of NBDE tests can be obtained from senior students who have already taken the exam, from your dental school library, or can be purchased directly from the ADA. Commercial test-taking agencies also offer question banks and test-taking simulations.

After taking a practice test always try to identify areas or concepts that are particularly troublesome. The identification of missed facts alone is not enough. There is the tendency to try and memorize the answers to questions you miss. This will prevent you from answering other similar questions correctly when the fundamental concept has not been learned. You will also want to evaluate why you are getting questions wrong. Perhaps you are not thoroughly reading through questions. Also, use practice tests to help gauge your test-taking skills. Analyze your time-management abilities.

You should not be discouraged if you are making a lot of mistakes on practice tests. Instead, you should use these opportunities to constantly motivate yourself to prioritize and reprioritize your study plan.

Also, use practice tests to help yourself with time-management so that you pace yourself accordingly during the exam. Try to spend equal time on all questions and try not to get bogged down on one question at the expense of others.

Texts, Syllabi, and Notes

Textbooks tend to be too detailed for high-yield review. Textbooks are helpful, however, if there are concepts that you have not fully grasped. Reading a paragraph or chapter from a textbook can help bring clarity to a concept that was not completely evident after lectures and coursework. Syllabi from individual faculty members may not correspond to and adequately reflect the material covered on the NBDE Part II. Similarly class notes may not adequately cover all the topic material that is covered on the boards. However, certain class notes may have been particularly helpful for understanding/memorizing certain concepts. These notes can be particularly valuable when you reach those particular sections in a more comprehensive review guide.

► TEST-TAKING STRATEGIES

Pacing

The exam is a one and a half-day exam. You have 7 hours on the first day to complete approximately 400 questions and 3 1/2 hours on the second day to complete 100 questions. On the first day, the day is split in half, so you will have 3 1/2 half hours to complete the first 200 questions, followed by a 1-hour lunch, and then another 3 1/2 hours to complete the second 200 questions. This works out to about 1 minute per question. On the second day, you will have 3 1/2 hours to complete 100 case-based questions. This works out to approximately 2 minutes 6 seconds per question. However, a considerable amount of time will be required to read the case synopsis, look at radiographs and clinical pictures, and to go over dental charting. Therefore, it may be more sensible to divide up the time in terms of number of case problems (usually 8–10). This works out to roughly 21–26 minutes per case. Whichever method you decide to use, make sure to practice your pacing method on several practice tests. An error in pacing can hurt your chances of doing well on the exam regardless of how much you prepared for the exam. Therefore, always be aware of how time has elapsed!

The following table adapted from ADA.org gives a breakdown of the NBDE Part II testing process:

Day 1	
Sign In	Tutorial
3½ hours	Approximately 200 randomly ordered disciplined-based test items
Maximum 1-hour lunch break	
3½ hours	Approximately 200 randomly ordered disciplined-based test items
Day 2	
Sign In	
3½ hours	100 patient case-based problems

Part II examinees must attend all three testing sessions.

Dealing with Each Question

Several techniques can be employed when dealing with each question. The most common technique is to read the question stem, think of the answer, and then consider the answer choices. A second technique is to look at the last sentence of the question stem, then skim through the answer choices, and then skim through the question stem extracting only pertinent information. The key is to try a variety of techniques during your practice sessions to see which method is most comfortable for you.

Difficult Questions

There will be several questions which appear workable but may take more time than the 1 minute per question time allotment you've given yourself. It can be tempting to try to figure out these questions for an excessive amount of time. However, every effort should be made to resist this temptation and to refocus on budgeting your time appropriately and spending time on questions that will not need as much effort. Difficult questions should be answered with your best guess and then marked for future consideration if you have enough time at the end of the test. This will help ensure that you spend adequate and equal time on all questions, and diminish the risk of missing questions.

Some test questions are experimental or may be incorrectly phrased. There may also be questions that are embedded into the exam as pretest items that do not count toward your final score. Therefore, do not dwell too long on any one question. They may not even count!

Guessing

Do not forget that there is no penalty for answering a question incorrectly. Therefore, don't leave any questions unanswered. Having at least a hunch is better than randomly guessing. If you don't even have a hunch, we recommend choosing the answer choice that you recognize over answer choices that are completely unfamiliar to you.

Changing Your Answer

Studies show that if you change your first answer to a question, you are two times as likely to change your answer from a incorrect answer to a correct answer than you are to change a correct answer to an incorrect answer. Therefore, if you have a strong reason to believe to change your answer, then it may be statistically in your favor to change your answer.

Avoiding Burnout

Frequent practice will help you to prevent burnout and to develop endurance. It may help to practice taking a test for 1/2 hour, then 1 hour, and then to work up to 3 1/2 hours. During the actual exam, you may begin to feel frustrated at some point. Don't give up. Try taking a small break for 30 seconds and then returning to the test after the brief breather. Extra time at the end of the test can be used to review marked questions or to recheck your answers. Rechecking answers you are very confident in can sometimes be just as important as reviewing questions that you are unsure about. Often, answers that you are confident in may be incorrect because you read the question stem incorrectly. Identifying these errors can be very valuable since you have the knowledge, and it is a simple matter of applying it correctly.

Testing Agencies

Joint Commission on National Dental Examinations
American Dental Association
211 E. Chicago Ave.
6th Floor
Chicago, IL 60611
312-440-2500
800-232-1694

Thomson Prometric
3110 Lord Baltimore Drive
Suite 200
Baltimore, MD 21244
www.prometric.com

CHAPTER 1

Operative Dentistry

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► DENTAL CARIES

Perhaps the most common chronic disease in the world, an infectious microbiologic disease of the teeth that results in localized demineralization and destruction of the calcified tissues, leading to loss of tooth structure.

Etiology

Carious lesions occur when a mass of bacteria adhere to the tooth surface (dental plaque) → plaque bacteria feast on refined carbohydrates → metabolize the sugars and produce acid byproducts → the acid lowers the pH of the plaque adherent to the tooth → the critical pH is reached at which demineralization of the adjacent tooth takes place.

- **Critical pH:** 5.5 (Some sodas have a pH of 3.5.)
- **Nonspecific plaque hypothesis:** All plaque cause caries.
- **Specific plaque hypothesis:** Only those that cause caries are pathogenic.
- **Fluoridation:** It has significantly decreased the number of caries, especially in children.
- **Saliva is carioprotective:** It acts as a buffer, carries minerals important for remineralization (Ca, PO₄, and F), has antimicrobial properties, and washes away food.

PAIN THEORY

There have been different thoughts on the production of pain secondary to carious or other insult. The current accepted school of thought is the *hydrodynamic theory*.

- When the tooth is subjected to insult, fluid movement through the tubules increases and the greater flow deforms the nerve endings in the pulp leading to pain response.
- Cold conductivity increases both the volume and the flow in the tubules resulting in pain stimulus.

**Cariogenic bacteria =
SALIVA
S. mutans, sanguis,
mitis, salivarius
A. viscus
Lactobacilli
Veillonella
A. naeslundii**

Responsible Organisms

- *Streptococcus mutans* and *Lactobacilli* are the most common cariogenic bacteria in coronal caries.
- *Actinomyces viscus* is the most common cariogenic bacteria in root surface or smooth surface caries.
- Dental plaque organisms—*Streptococcus sanguis* found earliest
- Other offenders: *Actinomyces naeslundii*, *Veillonella*, *Streptococcus salivarius*

Pathogenesis

ENAMEL CARIES

Caries in enamel have different properties than those in dentin.

- Acidogenic or physiochemical progression of structural destruction of enamel → demineralization due to lowered pH.
- Early lesions are capable of remineralizing or arresting, if pH is in favor of building and mineral content like fluoride is abundant.

- *Incipient caries* describes caries that have not progressed farther than enamel, they are reversible or able to remineralize.
- *Frank caries* describe caries that have progressed just into the dentinoenamel junction (DEJ).

PIT AND FISSURE CARIES

The shape of pits and fissure make these caries the most prevalent variant.

- Mostly *S. sanguis* and other strep.
- Narrow at the enamel surface and spreads wide at the DEJ (inverted V).
- Rapid destruction as many dentinal tubules are involved and undermining takes place.
- Actual lesion is often much larger than clinically presentable.
- Lesion progression parallels enamel rods.
- Prevent with fissurotomy and sealant.

SMOOTH SURFACE CARIES

Interproximal or cervical are the second most prevalent of all caries and are usually found just gingival to the proximal contact.

- Start wide at the surface and converges toward the DEJ (V shape).
- Slower progression as less tubules are affected and undermining is less.
- Prevent with fluoride.

ZONES OF CARIOUS ENAMEL

Also referred to as “zones of incipient lesion,” four zones have been characterized in a sectioned incipient lesion.

1. Translucent zone → the deepest zone, is termed accordingly due to its absent or composition-less appearance seen under polarized light.
2. Dark zone → represents remineralization and is termed so due to its inability to transmit polarized light.
3. Body zone → the largest zone, represents a demineralizing phase.
4. Surface zone → outermost zone, seems unaffected by the caries.

DENTIN CARIES

Acidogenic progression to the dentin layer results in a different pattern of destruction than that of enamel. The structure of dentin contains less mineralized tissue and instead more tubular structures, which allows for spread of the acidogenic destruction.

- Faster progression than enamel caries, because less mineral content.
- V-shaped caries with broad base at DEJ and the apex toward the pulp.
- Infected dentin → tubules are infected with many acid-producing bacteria, and acidogenic and proteolytic activity result in degradation.
- Affected dentin → bacteria present, but in smaller amounts. Demineralization occurs but still can be reversed if favorable environment assumes and infected layer is removed.



The DEJ provides the least resistance to caries and allows for rapid spread once approximated.

ZONES OF CARIOUS DENTIN

Five zones have been characterized in carious dentin from innermost to outermost. Only the first three zones are capable of remineralization. Zone 2 and 3 are termed *affected* and 4 and 5 are *infected*.

1. Normal dentin → no bacteria or byproducts present in this deepest unaffected area.
2. Subtransparent dentin → demineralization from acidogenesis, but no bacteria found in dentinal tubules.
3. Transparent dentin → softer than normal, further demineralization—yet still no bacteria found.
4. Turbid dentin → the zone of bacterial invasion: bacteria present in the dentin tubules. Must remove this zone.
5. Infected dentin → many bacteria found in this outermost carious zone. Must remove dentin to treat successfully.

ROOT SURFACE CARIES

Destruction of cementum and radicular or root dentin is more often found in older individuals (senile caries) with clinical recession and resultant exposure to plaque and acid-producing bacteria.

- Occurs when the root has been exposed to the oral environment and contaminated with plaque.
- Cementum surface is rougher than enamel and has greater mechanical advantage of acquiring plaque.
- Often associated with decreased salivary flow and decreased ability to provide adequate hygiene as seen in aging population.
- Spreads shallow along the surface, is ill-defined as characterized by a U shape.
- The relatively thin cementum provides little resistance to attack and results in a rapid progression, and progresses more rapidly than other lesions.
- The pathway of initial demineralization is along the Sharpey's fibers of the cementum.
- The shallow nature of these lesions lend to the finding that many of these lesions are associated with remineralization. Generally very dark lesions have been remineralized to some degree.
- Often asymptomatic.
- Difficult to restore.

RESIDUAL CARIES

Infected or cavitated tooth structure remaining after attempted removal in a completed cavity preparation.

- Can be intentional such as in indirect pulp capping procedures.

RECURRENT CARIES

Also known as secondary caries, is decay that remains in a completed cavity.

- Radiolucent bases or liners can be mistaken for recurrent decay on radiographic presentation.

RAMPANT CARIES

Rapidly progressing wide-spread caries are often the result of histological disadvantages, poor hygiene, drug abuse, radiation, high-sugar diets, or decreased salivary conditions.

- Acute onset
- Often associated with pain
- Deep and narrow presentation result in large cavitation

ARRESTED CARIES

Describes lesions that have remineralized.

- Hard
- Black or brown discoloration, a result of trapped debris and metallic ions
- Asymptomatic

► EXAMINATION AND DIAGNOSIS

The successful diagnosis of caries involves a good history, risk assessment, clinical evaluation, and radiographic analysis. The goal of examination is to diagnose caries activity before it becomes obvious on presentation. If found in premature or even incipient stages, a treatment approach to arrest and remineralize the faulty tooth structure is optimal. Only when changes are irreversible must we intervene with the infamous “drill and fill.”

- A good history and caries risk assessment is important and can determine the course of action taken; for example, a small carious lesion in a low-risk patient may not need any restoration, whereas the same lesion in a high-risk patient may necessitate one.
- Risk factors include history of prior caries, frequent sucrose intake, low fluoride intake (communities with nonfluoridated water), young or old age, poor oral hygiene, existing restorations, high concentrations of cariogenic bacteria, and salivary deficits. Table 1–1 shows risk factors for caries. Table 1–2 shows common drugs that affect salivary function.



The most common etiology of decreased salivary function is side effect to medications.

TABLE 1 – 1. Caries Risk Factors

Moderate Risk Factors	High Risk Factors
Exposed roots	Visible cavitation
Deep pit and fissures	Restoration in past 3 years
Interproximal enamel lesions	Visible plaque
Other white spots or discolorations	Frequent between meals snack (>3/day)
Recreational drug use	Inadequate saliva
	Appliances (ortho, retainer, removable partial dentures [RPD])

TABLE 1 – 2. Common Drugs That Reduce Salivary Function

MEDICATIONS	COMMON EXAMPLES
Anticholinergics	Atropine, glycopyrrolate, scopolamine
Diuretics	Hydrochlorothiazide
Local anesthetics (can temporarily aid during dental treatment)	Lidocaine
Antipsychotics	Chlorpromazine
Beta-blockers	Atenolol
Benzodiazepines	Alprazolam
Antihistamines	Chlorpheniramine

- Diagnosis of caries is dependent on multiple analysis, clinical and radiographic, and must take into account risk assessment. No single test is diagnostic unless grossly obvious.

Clinical Examination

Includes a look at the gross oral environment and individual teeth and surfaces.

- The following should be evaluated before looking at individual teeth and surfaces. Saliva quantity and quality, plaque, oral hygiene, gross caries, and existing dental work.
- Visual evidence of caries include cavitations, roughened or irregular appearances, color changes, and opacifications and/or translucency.
- Opacifications → initial demineralized enamel appears chalky or opaque when dried with air, and disappears when wet with water or saliva.
- Color changes → brown-gray discoloration indicates lateral spreading and undermined enamel. The discoloration seems to radiate away from the pits and fissures in a diffuse manner.
- Transillumination with a dental curing wand can reveal changes.
- A discolored marginal ridge may indicate proximal caries.
- Tactile evidence includes—surface roughness, softness, multiple “catches” with an explorer.
- Dark spots that are hard to tactile sensation resemble arrested lesions that have remineralized (like scar tissue of the teeth) especially in older individuals.
- To view proximal smooth surface caries temporary mechanical separation of the teeth helps with visualization and access for tactile instrumentation.
- Bacterial counts or salivary analyses → high quantities of colony-forming units (CFU) may be a helpful adjunctive diagnostic tool.

Criteria for diagnosis of pit and fissure caries =
FLOSS
Flakiness of enamel
Loss of translucency
Opaicity
Stickiness
Softening of the base of pit and fissure

Caries Detectors

Caries detectors can be used to visualize infection of tooth structure.

- A colored dye in an organic base adheres to the denatured collagen.
- Distinguishes between infected and affected dentin.

Radiographic Evaluation

Bitewing radiographs are the preferred view for caries detection, though occlusal caries are better detected with a panoramic view.

- Caries *cannot* be diagnosed radiographically without clinical examination.
- Pit and fissure caries appear as radiolucent areas that spread laterally under the occlusal surface.
- Bitewing radiographs are important in diagnosing proximal caries in tight contacts that limit access to visual or tactile evaluation.
- Radiographs underestimate the actual extent of caries—for example, a proximal lesion that appears to be two-thirds or more through the enamel has most likely penetrated the DEJ.
- Radiolucent lesions in the proximal surface that appear to be in enamel only and have no clinical cavitations should be considered reversible.



An explorer catch alone is not indicative of caries, and often explorer instrumentation is not recommended because it may result in iatrogenic cavitations.

► CAVITY PREPARATION

The surgical removal of carious tooth structure to prepare a cavity capable of retaining a dental material that will restore function, form, and esthetics. The following describe basic principles to cavity preparation for amalgam restorations.

- All prepared walls and internal line angles should be placed in dentin that is free of any destruction or infection and is hard and cannot be easily flaked away (sound dentin).
- The prepared cavity should be extended to include all decay and provide convenience for restoring and finishing the cavity.
- Enough depth and width should be prepared to prevent fracture of the tooth and restoration.
 - About 1/5 of the distance between buccal and lingual cusp tips.
 - At least 0.5 mm into the dentin.
 - Pulpal floor should be flat and parallel and perpendicular to the occlusal surface.
 - Line angles should be rounded and defined. Sharp line angles increase the risk of fracture.

Cavity Classification

Cavities and preparations can typically be classified in three ways:

1. Type of surface involved—pit and fissure vs. smooth surface.
2. Number of surface involved.
3. G.V. Black classification—most commonly used.

NUMBER OF SURFACES INVOLVED

- Simple cavity
 - Lesion is confined to one surface. Example: Occlusal lesion only.
- Compound cavity
 - Two surfaces of the same tooth. Example: Occlusal and buccal lesion.
- Complex cavity
 - Greater than two surfaces on same tooth.

G.V. BLACK CLASSIFICATION

Describes cavity preparations for amalgam restorations.

- **Class I** Lesion and required preparation is limited to the occlusal surface and involves pit and fissure caries.
 - Occlusal surface of posterior teeth.
 - Lingual surface of anterior teeth.
 - Difficult to see radiographically.
 - Mesial and distal walls should diverge occlusally.
 - Buccal and Lingual walls should be parallel to each other and perpendicular to the occlusal surface.
 - If the width of the marginal ridge is less than 1.6 mm, the preparation should be converted to a class II.
 - Retention is established by parallel walls and defined line angles.
- **Class II** Proximal caries on posterior teeth.
 - Can include both mesial and distal sides (MOD).
 - The occlusal outline form at the proximal segment is dictated by the position of the proximal contact and the extent of the carious lesion.
 - The proximal walls are divergent occluso-lingually.
 - The gingival floor should parallel the enamel rods.
 - Place a wedge between two contacting teeth when preparing and restoring the proximal segment. Wedges:
 - depress the gingiva apically
 - cause minimal separation
 - minimize oozing of fluids through the rubber dam
- **Class III** Cavities of proximal surfaces of anterior teeth that *do not* include the incisal angles.
 - Can be prepared and restored from a lingual or labial approach, and is dependent on the size and location, access, and esthetics.
 - For esthetic reasons unsupported enamel may be left intact.
 - Composite is the preferred material for class III and IV cavities that are in the esthetic zone (usually mesial to the distal contact of the canines).
- **Class IV** Cavities of proximal surfaces of anterior teeth that *do* include the incisal angles.
- **Class V** Cervical caries; in the gingival 1/3 of buccal or lingual surfaces.
 - Dictated by the extent of the carious lesion or denatured tooth structure.
 - Mesial and distal walls parallel the enamel rods direction.
 - The occlusal wall is longer than the cervical wall in ideal preparations, allowing for a trapezoidal shape.

The knowledge of dental instruments is of absolute importance, as the variety, use, and application dictate the success of operative treatment.

Hand Cutting Instruments

Make up most of the instruments used in finishing and restoring operative procedures.

- Made up of three standard components:
 - Handle → AKA shaft; can be single or double ended. Is using the part of the instrument that is held.
 - Shank → connects the blade to the handle; may be straight or angled.
 - Blade → carries the functional end (cutting edge or nib).
- Nib → working end of a blade or instrument that can be used for cutting, condensing, burnish, insert, and finish restorations.
- Cutting instruments are designated by using a formula (G.V. Black instrument formula) that describes the working end and the angle of the shank.
 - Width of the blade.
 - Angle of the primary cutting edge to the blade or plane of the instrument when the cutting edge is at an angle $<$ or >90 degree this fourth unit is added and it is placed in the second position of the formula. For example 15-85-8-12.
 - Length of the blade.
 - Angle of the blade → when the cutting edge.

CLASSIFICATION

Hand cutting instruments are described by four divisions: order, suborder, class, and subclass.

ORDER

Describes the purpose of the instrument; cutting instruments are termed *excavators*. Their function is to:

- Remove caries.
- Refine the anatomy of the preparation.

SUBORDER

Describes position and technique.

CLASS

Describes form and shape → the different types of cutting instruments.

- Chisel Family → The cutting edge makes a 90 degree angle with the plane of the blade, and has a blade that ends in a one-sided bevel.
 - Chisels are used to plane and cleave **enamel**.
 - They include chisels, hoes, angle formers, discoids, and cleoids.
 - Discoids and cleoids are now used primarily for carving amalgam.

The Chisel family named their son **CHAD**
Chisels
Hoes
Angle formers
Discoids and Cleoids

The Hatchet Family named their son **HEG**
Hatchets
Excavators
Gingival Marginal Trimmers

- Hatchet Family → The cutting edge is parallel with the plane of the instrument and has a blade that ends in a cutting edge in the plane of the handle.
 - They include hatchets, excavators, and gingival marginal trimmers (GMT).
 - GMT are used in planing gingival cavo surface margins and beveling axio-pupal line angles.
 - Comes in mesial and distal GMT.

SUBCLASS

Describes the angle of the shank → straight, mono (1), bin(2), or triple(3) angle.

► **RESTORATIONS**

Dental Materials and Biomaterial Science

When selecting a material to use in restoring lost tooth structure many factors must be considered in relation to the type of cavity and the appropriate material properties. The following physical and biological properties are used to discuss and compare the different dental materials.

ULTIMATE STRENGTH

Can be characterized by three variables—compressive, tensile, and shear—and is defined as the point on the stress-strain curve at which fracture occurs. Materials often differ in their types of strength; for example, amalgam has more compressive than tensile strength. Table 1–3 summarizes these differences in common dental materials.

- Compressive strength: stress required to fracture a material when forces are applied opposite and toward each other (pressing).
- Tensile strength: forces are applied opposite and away each other (pulling).
- Shear strength: forces are applied opposite and toward each other but at different positions (sliding).

TABLE 1 – 3 . Ultimate Strength of Common Dental Materials

MATERIAL	TENSILE STENGTH (MPa)	COMPRESSIVE STRENGTH (MPa)
Dentin	100	300
Enamel	10	400
Amalgam	50	400
Composite	40	300

TABLE 1-4. Thermal Expansion of Common Dental Materials

MATERIALS	COEFFICIENT OF THERMAL EXPANSION (PPM/°C * 10)
Tooth	10
Amalgam	20
Composite	30
Gold	10

DIMENSIONAL CHANGE

The percent of expansion or contraction of a material. In restorative dentistry this is most often studied as thermal expansion as the oral environment is subject to varying temperatures (drinking hot tea or iced tea). When the thermal expansion of tooth does not equal that of the restoration the differing expansions result in *leakage* of fluids between the two. *Percolation* refers to intermittent inlet and outlet of fluid leakage and can result in marginal decay. Of importance is the coefficient of thermal expansion which measures per unit length expansion if the material is heated by 1°C. Table 1-4 shows values of thermal expansion of common materials.

THERMAL CONDUCTIVITY

The number of calories or quantity of heat transferred per second across an area (cm²) and a length (cm) when the temperature difference is 1°C/cm. In English that means the ability of an object to transfer heat. For example if you heat a frying pan and touch it with your fingers, you will appreciate the pans high thermal conductivity, and can further appreciate that metals or alloys have higher thermal conductivities than composites. This also explains the use of liners and bases to protect the pulp when metals are used. Table 1-5 compares thermal conductivity of the common materials.

TABLE 1-5. Thermal Conductivity of Common Dental Materials

MATERIALS	THERMAL CONDUCTIVITY (CAL/SEC/CM ² [°C/CM])
Tooth	.002
Amalgam	.05
Composite	.002
Gold	.7

STRESS

Force per unit area or stress = force/area. Simple physics, for a given force the smaller the area for which it is applied, the greater the stress experienced. Thus if a restoration is built with sharp contacts, the small area is subject to greater stress than would be a broad contact with a larger area.

STRAIN

The change in deformation per unit length of a material subjected to stress. You can appreciate strain if you visualize the same stress applied to rubber vs. gold. Rubber would have far more deformation and therefore greater strain than gold.

ELASTIC MODULUS

The measure of stiffness or rigidity of a material, or the ratio of stress to strain below the elastic limit. In other words, the higher the elastic modulus, the more stiff or rigid is the material.

- *Elastic limit* is the greatest stress an object can be subjected to in which it can return to its original dimension once the force is removed.
- Gold → enamel → amalgam → composite → dentin.

PROPORTIONATE LIMIT

The stress at which the material no longer functions as elastic. It is the greatest stress that can be produced before permanent deformation exists. Yes for all practical purposes this is the same as elastic limit.

- *Yield strength* is an arbitrary stress point immediately higher than the proportionate limit and defines the point at which permanent deformation takes place or begins.
- Gold → enamel → amalgam → composite → dentin.

AMALGAM

A commonly used restorative material composed of a mixture of dental alloy and mercury. Although more esthetic materials like composite or porcelain are becoming more common, the favorable properties and low cost of amalgam have kept amalgam restorations in use today. Furthermore, amalgam is easy to use, and has proven longevity and versatility. Contrary to adversary belief, amalgam is safe, and the release of mercury vapor from existing restorations is, by current study, insignificant.

COMPOSITION AND PROPERTIES

Dental amalgam alloys consist mostly of silver (Ag) and tin (Sn) and copper (Cu), zinc (Zn), and or mercury (Hg) are usually present in small amounts. Varying the composition determines the physical properties of the amalgam. Table 1–6 summarizes the effect of each component on various physical properties of amalgam.

TABLE 1-6. Amalgam Composition and Properties

BY ↑	WORKING TIME AND CREEP	EXPANSION	STRENGTH	OTHER PROPERTIES
Ag	↓	↑	↑	
Sn	↑	↓	↓	
Cu	↓	↑	↑	↓corrosion
Hg	↑	↑	↓	Reacts to produce different phases
Zn		↑↑ when contaminated with saliva		Prevents oxidation by acting as a scavenger

STRENGTH

The final amalgam restoration should be strong enough to withstand everyday occlusal forces.

- Amalgam has 10 more compressive strength than tensile strength.
- Weak condensation during packing ↑voids resulting in a *weak* amalgam.
- Improper mixing → weak amalgam.

DIMENSIONAL CHANGE

Net contraction or expansion. For obvious reasons, a good amalgam restoration should not contract or expand after setting.

- Amalgam has a σ of thermal expansion 2 that of teeth.
- Properties that ↑setting expansion are more free mercury, shorter trituration time, small condensation pressure, and increase particle size.

CREEP

Describes the gradual, time-dependent, dimensional change that results from constant stress.

- The best contribution of copper to dental alloys is elimination of γ_2 and a consequent decrease in creep.

TARNISH AND CORROSION

Tarnish and corrosion are results of chemical reactions between amalgam and the local environment.

- Tarnish can result in discoloration and occurs at the amalgam surface.
- Corrosion can result in destruction of the restoration as it occurs deeper in the body of the amalgam.



Don't confuse thermal expansion with thermal insulation: Amalgam has a favorable coefficient of thermal expansion, but is a poor thermal insulator.



"High-copper alloys"—consist of 10–30% Cu by weight. Copper adds strength by eliminating the γ_2 phase (Sn_3Hg)



If amalgam is contaminated with moisture during condensation H_2O reacts with zinc to form H_2 gas, leading to greater expansion, decreased compressive strength, postop pain, and $\uparrow$ corrosion.

- The smoother, more well polished an amalgam the $\downarrow$ corrosion and tarnish.
- Marginal leakage $\downarrow$ with time because of $\uparrow$ corrosion byproducts: tin sulfide.
- Discolored, corroded, superficial layer on amalgam surface = sulfide.

TRITURATION

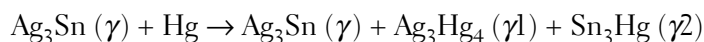
The process of mixing the alloy with mercury in the amalgamator.

- Undertrituated = dull, crumbly, $\downarrow$ strength, $\uparrow$ creep.
- Overtrituated = wet, runny, sticky, $\downarrow\downarrow$ strength, $\uparrow$ corrosion, $\downarrow$ setting expansion time, $\uparrow$ creep.
- Properly triturated = shiny, smooth, and homogenous.

The silver alloy is a fine powder that is composed mostly of Ag and Sn, and lesser amounts of elements like Cu and Zn. When this powder is mixed with Hg (in the amalgamator), dental amalgam is formed by a chemical reaction. The particles can be round (spherical), irregular (lathe-cut), or a mixture of the two (admixed alloy) (Table 1–7). Most practitioners use either spherical or admixed amalgam.

AMALGAMATION

The reaction of silver alloy with mercury.



- γ = strongest phase.
- γ_2 = weakest and most corrosive phase.
- Notice that the strongest phase has no mercury. High mercury content in amalgam restorations is detrimental to strength and other properties including marginal breakdown, fracture, and corrosion.
- You can minimize the amount of mercury-rich matrix by good condensation and carving, as the mercury-rich matrix will come to the surface and be removed.

Direct Esthetic Materials

COMPOSITE

Most commonly used direct esthetic material, because of their esthetic and wear resistant properties. They are often the material of first choice for anterior

TABLE 1–7. Properties of Amalgam Based on Alloy Shape

SILVER ALLOY SHAPE	ABILITY TO CARVE	WORKING TIME	Hg %	CONDENSATION FORCE NEEDED
Spherical		Faster	Less	Less
Admixed	Easier			More
Irregular			More	

class III, IV, and V restorations and have found increasing use as posterior class I and II restorations because of their favorable color properties. Furthermore, the thermal conductivity is much lower in composites when compared to amalgam or other metals and closely resembles that of natural teeth resulting in better thermal protection of the pulp.

COMPOSITION AND PROPERTIES

Composites consist of three phases: resin matrix, filler particles, and coupling agent. Initiators, accelerators, and pigments are needed to complete the final composite product.

- Filler particles → Fine (0.5–3 μm): crystalline silica (quartz), lithium aluminum silicate, glasses.
 - Microfine (0.04 μm): colloidal silica.
 - Microhybrid: contain both fine and microfine particles.
- Matrix → Bis-GMA (dimethacrylate), UDMA (oligomers).
- Coupling agent (Silane).
- Initiators → benzoyl peroxide, diketone, camphorquinone.
- Accelerators → organic amines.
- Pigments → inorganic pigments to provide 10 or more shades ranging from yellow to gray.



Quartz and lithium aluminum silicate fillers are not radiopaque and therefore should be avoided in use of posterior restorations, as they may resemble carious lesions on radiographs.

ACTIVATION

Can be either by chemical cure, light cure, or dual cure.

- Light activated: most common system used, the composite is polymerized by an intense blue light. The system uses a diketone which absorbs the light and an amine activator to initiate the polymerization process by supplying free radicals.
 - → 20–40 seconds of light to polymerize—darker shades require longer exposure time.
 - → Light source is a quartz-halogen bulb.
 - → Light is absorbed at 470 nm (blue light).
- Chemically activated: self-cure system, uses an organic peroxide initiator (benzoyl peroxide) and tertiary amine activator.

STRENGTH

Characterized by compressive and flexural strength, and hardness and wear.

- Microhybrid composites have ↑compressive and flexural strength than microfilled composites.
- ↑ filler volume = ↑hardness and wear.
- Composite restorations most likely fail in tension and bending.
- The bond between composite and etched tooth is primarily mechanical.

DIMENSIONAL CHANGE

Characterized by polymerization shrinkage and thermal expansion.

- Less resin = less shrinkage, therefore, microhybrid fillers have less shrinkage than microfilled composites.



The single most important factor in protecting the pulp from trauma, thermal, or chemical damage is dentin.

- Curing composite in layers ↓ shrinkage.
- ↑ resin matrix = ↑ σ of thermal expansion, “therefore,” ↑ in microfilled composites.
- Composite has higher thermal expansion than teeth and amalgam.

APPLICATIONS OF COMPOSITE

Composite is unique in its broad scope application, as different types of composite can be used for different restorations.

- Flowable composite: These have a low filler content (<50% by volume), are light cured, and are used for cervical lesions, small restorations, pediatric lesions, ad sealants.
- Condensable (packable) composite: Light activated, microhybrid fillers with >65% filler by volume. These are used in class I and II restorations.
- Core buildup composites: Usually tinted (blue) to distinguish from tooth structure. Self-cured or dual cured.

COMPOMERS

Composites modified with polyacid groups, used in low-stress-bearing areas.

- Less wear resistant than composite.
- Release fluoride.

GLASS IONOMERS

Fluoro-aluminosilicate glass powder and liquid solution of polymers and copolymers of acrylic acid, high-fluoride-releasing material used in class V restorations and low-stress-bearing areas in high caries risk individuals.

- Expansion coefficient similar to dentin.
- Low solubility.
- High opacity.

BONDING AGENTS

Are needed to provide an adequate bonding of composite to tooth structure.

- Three components: etchant, primer, and adhesive.
- Etchant = 37% phosphoric acid, removes smear layer.
- Primer and bond (adhesive) can be combined in one solution.
- Primer is a wetting agent and uses micromechanical and chemical bonding.
- Bonding agent is an unfilled resin, provides micromechanical retention.

Cements/Bases/Liners/Temporary Restoration

All of these represent the same materials and differ, principally, only in thickness:

- Base: 1–2 mm and replaces lost dentin structure beneath restorations.
- Cement: 0.5 mm and does as its name suggests, acts as a cement between tooth and restoration.
- Liners: 5 microns and protects the pulp.

CEMENTS

Hard brittle materials formed by a powdered oxide or glass + liquid. They are used to retain restorations on prepared teeth such as crowns, inlays, onlays, and veneers.

GLASS IONOMER

Most common water-based cements, mostly used for final cementation of crowns and bridges.

- Fluoride release, anticariogenic.
- Micromechanical and chemical bond.
- Nonirritating, though should use a CaOH base to protect pulp in deep cavity preparations.

ZINC POLYCARBOXYLATE

Water based, used mostly for final cementation of crowns and bridges.

- Nonirritating, not as strong as counterparts.
- Zinc oxide powder and *polyacrylic acid* water solution.

ZINC PHOSPHATE

Water based, used for final cementation, though not as popular as other cements.

- Highly acidic (initial pH 4.2); therefore, need a varnish to protect the pulp.
- Powdered zinc oxide + phosphoric acid liquid.
- Mechanical bond.
- Fast setting time, low solubility, low acidity of *set* cement.
- ↑ Powder: water, warmer slab, faster incorporation of powder to liquid = ↓ setting time.

ZINC OXIDE-EUGENOL (ZOE)

Oil-based, sedative effect of pulp, works well with exposed dentinal tubules.

- Cannot use with composite, because eugenol inhibits polymerization.
- Can be used as a temporary cement.
- Zinc oxide + rosin + zinc acetate + accelerator + eugenol liquid.
- Low strength.

BASES

Used to provide support and thermal protection for the pulp.

- Properties of bases include: high strength and low thermal conductivity.
- Glass ionomers → used as bases for posterior composites and porcelain or composite inlays and onlays, and amalgams.
- CaOH → used as bases for amalgam or composite restorations.
- Zinc phosphate or zinc polycarboxylate → used under gold restorations.
- 2° bases → zinc phosphate cement over CaOH base over a pulpal exposure (direct pulp cap).



*Eugenol inhibits the
polymerization of composite
resins.*

LINERS AND VARNISHES

Protective barriers, function to protect the pulp by providing therapeutic benefits, they are too thin to provide thermal insulation.

- Recently being replaced by dentin bonding agents.
- Liners: suspensions of Dycal (CaOH) in water, and may contain fluoride.
- Varnishes: solution liners of resin (Copalite) in liquid.
- Don't use varnishes with composite because may be disrupted by the monomers.

INTERIM RESTORATIONS

Cements mixed to a thick consistency, can be used as temporary fillings.

- ZOE—most commonly used (IRM)
- Protects the pulp, reduces pulpal inflammation, and maintains structure.

CHAPTER 2

Pharmacology

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Absorption

The process of drug uptake from the site of administration and transfer into the blood stream. The site of administration will dictate the rate and efficiency of absorption. Sites of pharmacologic administration include intravenous delivery (where absorption is total) and other sites, such as oral, intramuscular, and transmucosal/transdermal (where absorption is only partial).

Bioavailability

A measure of how much drug reaches the circulatory system and is available at the site of action. Factors influencing the bioavailability of a drug include:

- Route of administration.
- Degradation of drug prior to absorption.
- Gastrointestinal (GI) absorption mechanisms (e.g., active transport vs. passive diffusion).
- Solubility—very hydrophilic drugs unable to cross lipid-rich cell membranes and very hydrophobic drugs unable to be absorbed due to insolubility in aqueous fluids. To be well absorbed, has to be largely hydrophobic.
- Hepatic first pass effect.
- Drug chemistry—presence of binders or dispersing agents, particle size, and crystal forms.

Drug Distribution

The ability of a drug to move from the circulatory system into the interstitium and tissues. Factors include:

- Blood flow.
- Protein binding.
- Permeability—ability to cross capillary barriers and specific types of capillary barriers that are largely impermeable such as the blood brain barrier.

VOLUME OF DISTRIBUTION (V_D)

A theoretical amount of fluid the drug is dispersed in after administration.

$$V_D = \frac{\text{total amount of drug in the body}}{\text{blood concentration of drug}}$$

DRUG METABOLISM

- **Phase I reactions:** Convert molecules into often still active slightly polar, water-soluble metabolites through oxidation, reduction, or hydrolysis reactions (e.g., cytochrome P-450 system).
- **Phase II reactions:** Convert metabolites into inactive polar metabolites via acetylation, glucuronidation, or sulfation that are then excreted by the kidneys.

Some drugs undergo phase II reactions directly and some drugs undergo phase II reactions before phase I.

DRUG ELIMINATION

- **Zero-order elimination:** Elimination of drugs in a linear constant fashion regardless of concentration. Concentration will decrease linearly. Drug examples include alcohol, phenytoin, and aspirin (at high or toxic doses).
- **First-order elimination:** Elimination of drugs in a proportional fashion to drug concentration. Concentration will decrease exponentially with time.

PHARMACODYNAMICS

- **Agonist:** Drugs that bind to receptors and elicit a biologic response by stabilizing the receptors in their active conformation.
 - Full agonist: elicits a maximal response by activating all or a portion of the receptors.
 - Partial agonist: elicits a less than maximal response even if all the receptors are occupied.
- **Antagonist:** Drugs that block the normal physiologic function of a receptor.
 - Competitive antagonists compete with agonist for a receptor and can be overcome by increasing the concentration of the agonist. Shifts an agonist curve to the right.
 - A noncompetitive antagonist inhibits by causing irreversible changes to receptors. Shifts an agonist curve downward.

THERAPEUTIC INDEX

A comparison of the amount of an agent (e.g., drug) that causes a therapeutic effect to the amount of that same agent that causes toxic effects.

$$\text{Therapeutic index} = \text{LD}_{50}/\text{ED}_{50}$$

- LD_{50} = Lethal dose of a drug for 50% of population
- ED_{50} = Effective dose of a drug for 50% of population

PRESCRIPTION WRITING

All written prescriptions should include the following (see Fig. 2–1):

- Date of issue.
- Patient's full name, address, and date of birth.
- Prescriber's full name, address, telephone number, DEA number, and signature.
- Drug name, dose, form, and amount.
- Directions for use.
- Refill instructions.

DRUG EFFECTS

- **Tolerance:** The need to increase the dose in order to achieve the same effects originally achieved by a lower dose.

Dr. General Dentist
 555Amalgam Lane
 Composite, NY 12345
 Tel: 123-456-7890
 DEA# 1234567

Amoxicillin 500mg Tablets.
 Sig: Take 1 Tablet by mouth 3 times a day
 Disp: 21 Tablets.
 Refills: None

Signature:
 Date:

FIGURE 2 - 1 . Sample prescription.

- **Physical dependence:** Physiologic need for the substance when on cessation will illicit negative physical symptoms.
- **Drug abuse:** Use of illicit drugs or prescription or over-the-counter medications for purposes other than they are indicated for or in amounts greater than prescribed.

▶ AUTONOMICS

Direct Acting Adrenergics

Drug	Mechanism	Clinical Application
Epinehrine	$\alpha_1, \alpha_2, \beta_1, \beta_2$	Bronchospasm, glaucoma, anaphylactic shock, vasoconstrictor in anesthetics
Norepinephrine	$\alpha_1, \alpha_2, \beta_1$	Vasoconstriction, hypotension, shock
Isoproterenol	β_1, β_2	Bronchodilator
Dopamine	$D_1 = D_2 > \alpha_1, \alpha_2, \beta_1, \beta_2$	Shock
Dobutamine	$\beta_1 > \beta_2$	Congestive heart failure
Phenylephrine	$\alpha_1 > \alpha_2$	Nasal decongestion, pupil dilator
Methoxamine	$\alpha_1 > \alpha_2$	Paroxysmal supraventricular tachycardia, hypotension
Clonidine	α_2	Hypertension
Metaproterenol	β_2	Bronchospasm, bronchodilator
Albuterol	β_2	Bronchodilator
Terbutaline	β_2	Bronchodilator, reduce uterine contractions

Indirect Acting Adrenergics

Drug	Mechanism	Clinical Application
Amphetamine	Indirect acting general agonist	Narcolepsy, attention deficit disorder

Mixed Action Adrenergics

Drug	Mechanism	Clinical Application
Ephedrine	Indirect acting general agonist and direct α and β agonist	Asthma, nasal decongestion, hypotension

Nonselective α -Adrenergic Blockers

Drug	Clinical Application	Adverse Effects
Phenoxybenzamine (irreversible)	Pheochromocytoma	Postural hypotension, reflex tachycardia
Phentolamine (reversible)	Pheochromocytoma	Postural hypotension, reflex tachycardia

β_1 -Adrenergic Blockers

Drug	Clinical Application	Adverse Effects
Doxazosin Prazosin Terazosin	Hypertension, urinary retention	First dose postural hypotension, headache, dizziness

Nonselective β -Adrenergic Blockers

Drug	Clinical Application	Adverse Effects
Propanolol	Hypertension Angina pectoris Myocardial infarction Glaucoma Migraine	Bronchoconstriction, arrhythmias, sexual impairment, fasting hypoglycemia
Timolol Nadolol	Glaucoma Hypertension	More potent than Propanolol
Labetalol (with α_1 block)	Hypertension	Orthostatic hypotension, dizziness, bradycardia, fatigue, drowsiness

β_1 -Adrenergic Blockers

Drug	Clinical Application	Adverse Effects
Acebutolol Atenolol Metoprolol	Hypertension	Hypotension, bradycardia, fatigue, drowsiness

Agents That Affect Neurotransmitter Release or Uptake

- **Reserpine:** Blocks Mg^{++} /ATP-dependent transport of norepinephrine (NE), serotonin, and dopamine from the cytoplasm into storage vesicles in the adrenergic nerves.
- **Guanethidine:** Inhibits response of adrenergic nerves to stimulation by blocking release of NE.
- **Cocaine:** Inhibits the reuptake of NE by adrenergic neurons by blocking the Na^+/K^+ activated ATPase.

Cholinergic Agonists (Direct Acting)

Drug	Actions	Clinical Application
Bethanechol	Increases intestinal motility and stimulates detrusor muscle of bladder	Atonic bladder
Carbachol Pilocarpine	Produces rapid miosis and contraction of ciliary muscles	Glaucoma

Cholinergic Agonists (Indirect Acting)

Drug	Actions	Clinical Application
Edrophonium Neostigmine Physostigmine Pyridostigmine Echothiophate	Inhibits acetylcholinesterase increasing Ach in Neuromuscular Junction	Urinary retention Reversal of neuromuscular junction blockade Myasthenia gravis Glaucoma

Antimuscarinic Agents

Drug	Clinical Application	Adverse Effects
Atropine	Antispasmodic (GI and bladder) Mydriasis Antisecretory (eyes, airway) Antidote for cholinergic agonists	Dry mouth Blurred vision Tachycardia Constipation Disorientation
Scopolamine	Antimotion sickness Sedation	Similar to atropine
Ipratropium	Asthma Chronic obstructive pulmonary disease (COPD)	

Neuromuscular Blockers

Drug	Actions	Applications
Succinylcholine	Depolarizing neuromuscular junctional (NMJ) blockade	Rapid endotracheal intubation
Tubocurarine Atracurium Doxacurium Pancuronium	Nondepolarizing NMJ blockade	Adjuvant drugs in anesthesia for relaxation of skeletal muscles

Ganglionic Blockers

Drug	Actions
Hexamethonium	Nicotinic Ach receptor antagonist

► CENTRAL NERVOUS SYSTEM DRUGS

Anxiolytic and Hypnotic Drugs

BENZODIAZEPINES

Diazepam, Lorazepam, Clonazepam, Triazolam, Midazolam, Chlordiazepoxide, Alprazolam, Oxazepam

Action	Applications	Adverse Effects
Facilitates gamma-aminobutyric acid (GABA) receptor binding by increasing the frequency of chloride channel opening	Anxiety Sedation Muscle spasticity Seizures Sleep disorder Alcohol withdrawal	Drowsiness and confusion Dependence Potentiate alcohol effects and other central nervous system (CNS) depressants

Flumazenil: treatment of benzodiazepine overdose. Competitive GABA receptor.

Other Anxiolytics

Drugs	Actions	Applications
Buspirone	Appears to be mainly mediated through actions on serotonin receptors	Anxiety disorder
Hydroxyzine	Antihistamine	Antiemetic Anxiety disorder

BARBITURATES

Phenobarbital, Pentobarbital, Thiopental, Secobarbital

Action	Applications	Adverse Effects
Potentiates GABA receptor binding by increasing the duration of chloride channel opening	Anesthesia Seizures Anxiety	Drowsiness and confusion Dependence Potentiate alcohol and other CNS depressants Cardiovascular and respiratory depressant Drug interactions (induce cytochrome P-450)

NONBARBITUATE SEDATIVES

- **Chloral hydrate:** Used for short-term treatment of insomnia and as a sedative before dental or minor medical procedures. Rapidly metabolized into trichloroethanol and trichloroacetic acid by body. Cardiovascular and pulmonary depressant at high doses.
- **Antihistamines:** Diphenhydramine and doxylamine are effective in treatment of mild insomnia.

Anticonvulsants

Drugs	Mechanism	Adverse Effects
Carbamazepine	Block Na channels	Liver toxicity Blood dyscrasia Induction of cytochrome P-450
Phenytoin	Block Na channels	Gingival hyperplasia SLE-like syndrome Diplopia Ataxia Induction of cytochrome P-450
Gabapentin	Increases presynaptic GABA release	Ataxia Sedation
Valproic acid	Blocks Na channels and increases GABA concentration	Hepatotoxicity Neural tube defects
Benzodiazepines	Facilitates GABA binding	Sedation Dependence Tolerance

Antidepressants

SELECTIVE SEROTONIN REUPTAKE INHIBITORS

Paroxetine, Fluoxetine, Sertraline

Mechanism: Specifically inhibits serotonin reuptake

Application: Depression

Adverse effects: Anxiety, nausea, sexual dysfunction

TRICYCLIC ANTIDEPRESSANTS

Amitriptyline, Imipramine, Nortriptyline, Desipramine, Trimipramine, Doxepin

Mechanism: Inhibits reuptake of NE and serotonin into presynaptic nerve terminals

Application: Depression and panic disorders

Adverse effects: Antimuscarinic effects, postural hypotension, sedation, convulsions, arrhythmias

MONOAMINE OXIDASE INHIBITORS

Phenelzine, Isocarboxazid, Tranylcypromine

Mechanism: Increases monoamine (NE, serotonin, and dopamine) stores within the neurons and subsequent release of excess neurotransmitters into the synapse

Application: Depression, anxiety, and phobic states
Adverse effects: Drowsiness, postural hypotension, dry mouth, dysuria, and constipation

LITHIUM

Mechanism: Unknown
Application: Bipolar disorders
Adverse effects: Narrow therapeutic window, tremor, polyuria, and hypothyroidism

ANTIPSYCHOTICS (NEUROLEPTICS)

Haloperidol, Chlorpromazine, Thioridazine, Fluphenazine, Prochlorperazine
Mechanism: Block dopamine D₂ receptors
Application: Psychosis, acute mania, schizophrenia
Adverse effects: Extrapyramidal effects, tardive dyskinesia, antimuscarinic effects, postural hypotension

ANTIPSYCHOTICS (ATYPICAL)

Risperidone, Clozapine, Olanzapine
Mechanism: Block dopamine D₂ and serotonin (5HT₂) receptors
Application: Schizophrenia, mania, depression
Adverse effects: Lesser extrapyramidal and antimuscarinic effects than neuroleptics

► CARDIOVASCULAR

CARDIAC GLYCOSIDES

Digitoxin, Digoxin
Mechanism: Inhibits Na⁺/K⁺ ATPase of cardiac cell membranes resulting in increase Na⁺ concentration intracellularly. This favors the Na⁺/Ca⁺ antiport causing an influx in Ca⁺⁺ intracellularly ultimately resulting in increased contractility of the cardiac muscle
Application: Congestive heart failure
Adverse effects: Dysrhythmia, nausea, vomiting, blurred vision, digoxin toxicity (narrow therapeutic index)

ANTIARRHYTHMIC

Class I: Quinidine, Lidocaine, Procainamide, Flecainide, Disopyramide
Mechanism: Na⁺ channel blockers which can either slow phase 0 depolarization or shorten phase 3 repolarization of cardiac muscle cells

Class II: β -blockers (Propranolol, Metoprolol, Pindolol, Esmolol)

Mechanism: β -Adrenoreceptor blockers which can decrease phase 4 cardiac depolarization

Class III: Sotalol, Amiodarone, Bretylium

Mechanism: K^+ channel blockers causing a prolongation of phase 3 repolarization

Class IV: Calcium channel blockers (Verapamil, Diltiazem)

Mechanism: Block calcium channels resulting in a shortened action potential

ANTIHYPERTENSIVES

DIURETICS

Loop Diuretics (Furosemide)

Mechanism: Sulfonamide loop diuretic that inhibits the $Na^+/K^+/2Cl^-$ triple cotransporter of the thick ascending loop of Henle

Applications: Hypertension, edematous states

Adverse effects: Hyperuricemia, ototoxicity, hypokalemia, hypovolemia

Thiazide Diuretics (Hydrochlorothiazide)

Mechanism: Inhibits Na^+/Cl^- reabsorption in the distal convoluted tubules

Applications: Hypertension, congestive heart disease

Adverse effects: Hyperuricemia, hypokalemia

β -BLOCKERS

Propranolol, Metoprolol, Atenolol, Labetalol

Mechanism: Lowers cardiac output by decreasing sympathetic outflow from the CNS and inhibits the release of renin from the kidney

Applications: First-line drug therapy for hypertension

Adverse effects: Bradycardia, hypotension, insomnia, sexual dysfunction

α_1 -BLOCKERS

Prazosin, Oxazosin, and Terazosin

Mechanism: Competitive α_1 -adrenergic receptor blocker which causes a decrease in peripheral vascular resistance

Adverse effects: First dose postural hypotension, headache, dizziness

ACE INHIBITORS

Captopril, Enalapril, Lisinopril, Benazepril

Mechanism: Blocks angiotensin converting enzyme resulting in the decrease in conversion of angiotensin I into angiotensin II, potent vasoconstrictor

Adverse effects: Cough, hypotension, hyperkalemia, rashes, fever

Side effects of ACEIs
CAPTOPRIL
 Cough
 Angioedema
 Potassium excess
 Taste changes
 Orthostatic hypotension
 Pregnancy
 contraindication
 Rash
 Indomethacin inhibition
 Liver toxicity

ANGIOTENSIN II ANTAGONIST

Losartan

Mechanism: Angiotensin II receptor blocker

Adverse effects: Fetal toxicity

CALCIUM CHANNEL BLOCKERS

Verapamil, Diltiazem, Nifedipine

Mechanism: block L-type calcium channels in smooth muscle of the coronary and peripheral vessels and in the heart

Adverse effects: Dizziness, constipation, headache, fatigue, hypotension

ANTIANGINAL DRUGS

NITROGLYCERIN AND ISOSORBIDE DINITRATE

Mechanism: Nitric oxide release in smooth muscle cells cause an increase cGMP resulting in smooth muscle relaxation and vasodilation. Affects veins greater than arteries

Applications: Angina and pulmonary edema

Adverse effects: Dizziness, constipation, headache, fatigue, hypotension

β -BLOCKERS

Mechanism: Lowers cardiac output by decreasing sympathetic outflow and decreasing the workload of the heart. Reduces frequency and severity of angina attacks

ANTICOAGULANTS

HEPARIN

Mechanism: Binds and activates antithrombin III resulting in the inactivation of thrombin and factor Xa

Application: Pulmonary embolism, stroke, deep vein thrombosis. Follow PTT

Adverse effects: Bleeding, thrombocytopenia

Overdose Reversal: Protamine sulfate

WARFARIN

Mechanism: Inhibits the synthesis of vitamin K-dependent clotting factors: II, VII, IX, and X, as well as regulatory factors protein C and protein S

Application: Long-term anticoagulation. Follow PT/INR

Adverse effects: Bleeding, teratogenic

Overdose Reversal: Vitamin K

ANTHYPERLIPIDEMICS

HMG-CoA REDUCTASE INHIBITORS

Lovastatin, Pravastatin, Simvastatin, Fluvastatin

Mechanism: Competitively inhibit HMG-CoA reductase, the rate-limiting step in de novo cholesterol synthesis

Adverse effects: Rhabdomyolysis, myopathy, liver failure

Side effects and contraindications of HMG-CoA reductase inhibitors
HMG-CoA
Hepatotoxicity
Myositis
Girl in pregnancy/
Growing children
Coumadin/Cyclosporine interactions

▶ ANTIMICROBIALS

CELL WALL SYNTHESIS INHIBITORS

PENICILLINS

Penicillin V and Penicillin G

Mechanism: Binds penicillin binding proteins; blocks transpeptidase cross linking of bacterial cell wall; activates autolysins in bacterias. Not resistant to beta lactamase

Spectrum: Gram-positive cocci, gram-positive rods, gram-negative cocci, and spirochetes

Adverse effects: Hypersensitivity reactions, diarrhea, hemolytic anemia

NARROW SPECTRUM PENICILLINS

Methicillin, Dicloxacillin, Nafcillin

Mechanism: Same as penicillin except beta lactamase resistant because of bulkier R-group

Spectrum: *Staphylococcal aureus*

Adverse effects: Hypersensitivity reactions, nephritis

EXTENDED SPECTRUM PENICILLINS

Amoxicillin, Ampicillin

Mechanism: Same as penicillin but is beta lactamase resistant. Combined with beta lactamase inhibitors such as clavulanic acid (beta lactamase inhibitors) to enhance spectrum. Amoxicillin has greater oral bioavailability

Spectrum: Gram-positive bacterias such as enterococci and certain gram-negative rods such as *Haemophilus influenzae*, *Escherichia coli*, *Proteus mirabilis*, *Listeria monocytogenes*, *Salmonella*

Adverse effects: Hypersensitivity reactions, pseudomembranous colitis

ANTIPSEUDOMONAS PENICILLINS

Piperacillin, Carbenicillin, Ticarcillin

Mechanism: Same as penicillin but is beta lactamase resistant. Combined with beta lactamase inhibitors such as tazobactam (beta lactamase inhibitors) to enhance spectrum

Spectrum: Extended spectrum effective against many gram-negative bacilli and also against *Pseudomonas aeruginosa*

Adverse effects: Hypersensitivity reactions

CEPHALOSPORINS

- **Mechanism:** Beta lactam drugs that inhibit cell wall synthesis but are more resistant to beta lactamase.
- **First generation:** Cefazolin, cephalixin, cefadroxil. Effective against gram-positive cocci and also against *Proteus mirabilis*, *E. coli*, and *Klebsiella pneumoniae*.
- **Second generation:** Cefaclor, cefoxitin, cefuroxime, cefamandole. Has some decreased gram-positive activity but has increased gram-negative activity to three additional organisms (*Enterobacter aerogenes*, *H. influenzae*, and some *Neisseria* species).
- **Third generation:** Cefotaxime, ceftazidime, ceftriaxone, cefdinir, cefixime. Has some decrease gram-positive activity compared to first- and second-generation cephalosporin but has enhanced gram-negative activity against most other gram-negative enteric organisms and *Serratia marcescens*.
- **Fourth generation:** Cefepime. Increased activity against *Pseudomonas* and gram-positive organisms (*Streptococci* and *Staphylococci*).
- **Adverse effects:** Hypersensitivity reactions. Cross-hypersensitivity with penicillins occur in 5–10%. Disulfiram-like effect with cefamandole. Anti-vitamin K effects with cefamandole and cefoperazone causing bleeding.

CARBAPENEMS

Imipenem

Mechanism: Beta lactam drug that inhibits cell wall synthesis but are beta lactamase resistant

Spectrum: Broadest beta lactam antibiotic spectrum effective against penicillinase producing gram-positive organisms and gram-negative organisms, anaerobes, and *Pseudomonas aeruginosa*

Adverse effects: GI upset

MONOBACTAMS

Aztreonam

Mechanism: Beta lactam drug resistant to beta lactamase

Spectrum: Narrow spectrum primarily against *Enterobacteria*. Especially useful against aerobic gram-negative rods

Adverse effects: Skin rash and occasional abnormal liver functions. No cross-hypersensitivity reactions with penicillin

VANCOMYCIN

Mechanism: Inhibits synthesis of bacterial cell wall phospholipids by binding D-ala and D-ala portion of cell wall precursors. Bactericidal and resistance occurs with amino acid change of D-ala and D-ala to D-ala and D-lac.

Spectrum: Multidrug-resistant gram-positive organisms such as *Staphylococcus aureus* and *Clostridium difficile* (pseudomembranous colitis).

Adverse effects: Diffuse flushing (“red man syndrome”), nephrotoxicity, ototoxicity, phlebitis.

PROTEIN SYNTHESIS INHIBITORS

CLINDAMYCIN

Mechanism: Works at 50S ribosomal subunit by preventing peptide formation. Bacteriostatic.

Spectrum: Anaerobic organisms such as *Bacteroides fragilis* and *Clostridium perfringens*.

Adverse effects: Pseudomembranous colitis.

TETRACYCLINES

Tetracycline, Demeclocycline, Doxycycline, and Minocycline

Mechanism: Binds to 30S ribosomal subunit and blocks access of tRNA inhibiting bacterial protein synthesis. Avoid taking with milk antacids or iron containing compounds because divalent cations inhibit its absorption in the gut.

Spectrum: Wide spectrum active against *Vibrio cholerae*, acne, *Chlamydia*, *Ureaplasma urealyticum*, *Mycoplasma pneumoniae*, *Borrelia burgdorferi*, *Rickettsia*, and *Tularemia*.

Adverse effects: Discoloration of teeth and inhibition of bone growth in children. Photosensitivity, GI irritation, and fatal hepatotoxicity in pregnant women.

MACROLIDES

Azithromycin, Clarithromycin, and Erythromycin

Mechanism: Binds to 50S ribosomal subunit and inhibits the translocation steps of protein synthesis.

Spectrum: Upper respiratory infections, pneumonias, *Mycoplasma*, *Legionella*, *Chlamydia*, *Neisseria*, and gram-positive cocci (Strep infections in patients allergic to penicillin).

Adverse effects: Cholestatic hepatitis, GI irritation, ototoxicity.

AMINOGLYCOSIDES

Amikacin, Gentamicin, Neomycin, Netilmicin, Streptomycin, and Tobramycin

Mechanism: Binds to separated 30S ribosomal subunit interfering with assembly of functional ribosomal apparatus. Requires O₂ for uptake.

Spectrum: Aerobes only. Gram-negative rods and synergistic with beta lactam antibiotics.

Adverse effects: Teratogen, ototoxicity, nephrotoxicity.

CHLORAMPHENICOL

Mechanism: Binds 50S ribosomal subunit and inhibits protein synthesis at the peptidyl transferase reaction.

Spectrum: Broad spectrum antibacterial active against *H. influenzae*, *Neisseria meningitidis*, and *Streptococcus pneumoniae*. Also active against other organisms such as *Rickettsiae*.

Adverse effects: Hemolytic anemias and gray baby syndrome.

FLUOROQUINOLONES

Ciprofloxacin, Enoxacin, Levofloxacin, Norfloxacin, Ofloxacin, Trovafloxacin

Mechanism: Inhibits DNA gyrase (Topoisomerase II).

Spectrum: Gram-negative rods of GI and urinary tracts including *Pseudomonas aeruginosa*, *Neisseria*. Has some activity against some gram-positive organisms.

Adverse effects: Tendonitis and tendon rupture. GI irritation.

Antibiotic Prophylaxis Guidelines

2007 American Heart Association/American Dental Association antibiotic prophylaxis guidelines for infective endocarditis (adapted from Policy Guide for Consultation and Management of Medically Complex Patients—Department of Diagnostic Sciences Nova Southeastern University College of Dental Medicine)

PATIENTS REQUIRING PROPHYLAXIS

- History of prosthetic cardiac valve.
- History of previous infective endocarditis.
- History of congenital heart disease (CHD):
 - Unrepaired cyanotic CHD, including palliative shunts and conduits.
 - Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure.
 - Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization).
- History of cardiac transplantation recipients who develop cardiac valvulopathy.

Patients should be premedicated for all dental procedures that involve:

- manipulation of gingival tissue
- the periapical region of teeth
- perforation of the oral mucosa

The following procedures and events do not need prophylaxis even if the patient has one of the four cardiovascular diagnoses mentioned above:

- Routine anesthetic
- Injections through noninfected tissue
- Taking dental radiographs
- Placement of removable prosthodontic or orthodontic appliances

- Adjustment of orthodontic appliances
- Placement of orthodontic brackets
- Shedding of deciduous teeth
- Bleeding from trauma to the lips or the oral mucosa

DRUG OF CHOICE

Amoxicillin 2 g 30–60 minutes prior to dental procedure

IF ALLERGIC TO PENICILLINS OR AMPICILLIN

- Clindamycin 600 mg 30–60 minutes prior to the dental procedure
- Cephalexin 2 g 30–60 minutes prior to the dental procedure
- Azithromycin 500 mg 30–60 minutes prior to the dental procedure
- Clarithromycin 500 mg 30–60 minutes prior to the dental procedure

When necessary, antibiotics for prophylaxis should be administered in a single dose 30–60 minutes before the dental procedure. If the dosage of the antibiotic is inadvertently not administered before the procedure, the dosage can be administered up to 2 hours after procedure.

Antifungals

AMPHOTERICIN B

Mechanism: Binds to ergosterol in cell membrane of fungi and forms pores that allows electrolytes and small molecules to leak from the cell causing cell death.

Spectrum: Wide spectrum of systemic mycoses such as *Candida albicans*, *Histoplasma capsulatum*, *Cryptococcus neoformans*, *Coccidioides immitis*, strains of *aspergillus* and *Blastomyces dermatitidis*.

Adverse effects: nephrotoxicity, arrhythmias, fevers/chills.

NYSTATIN

Mechanism: Similar to amphotericin B

Spectrum: Oral candidiasis

Adverse effects: Nausea and vomiting

AZOLES

Fluconazole, Ketoconazole, Itraconazole, Miconazole, and Voriconazole

Mechanism: Blocks synthesis of ergosterol.

Spectrum: Systemic mycoses with same spectrum as amphotericin B with the addition of histoplasmosis.

Adverse effects: Endocrine effects (gynecomastia, impotence, menstrual irregularities, and decreased libido), liver dysfunction, fevers/chills.

FLUCYTOSINE

Mechanism: Inhibits DNA synthesis by conversion to fluorouracil and inhibiting thymidylate synthetase.

Spectrum: Systemic mycoses such as *Cryptococcosis*, *Candidiasis*, and *Chromoblastomycosis*.

Adverse effects: Bone marrow depression, hepatic dysfunction, and GI irritation.

GRISEOFULVIN

Mechanism: Interacts with microtubules to disrupt the mitotic spindle and inhibits mitosis.

Spectrum: Dermatophytes—*Trichophyton*, *Microsporum*, and *Epidermophyton*.

Adverse effects: Well tolerated.

Antivirals

AMANTADINE

Mechanism: Blockade of a viral membrane matrix protein, M2, which is required for fusion of the viral membrane with cell membrane. Prevents viral penetration and uncoating.

Spectrum: Influenza A; Parkinson's disease.

Adverse effects: Insomnia, ataxia, and dizziness.

RIBAVIRIN

Mechanism: Inhibits synthesis of viral mRNA by inhibiting synthesis of guanine nucleotides.

Spectrum: Broad spectrum of RNA and DNA viruses including respiratory syncytial virus and hepatitis C.

Adverse effects: Hemolytic anemia and teratogenic.

OSELTAMIVIR AND ZANAMVIR

Mechanism: Influenza neuraminidase inhibitors

Spectrum: Influenza A and B

Adverse effects: GI irritation

ACYCLOVIR

Mechanism: Phosphorylated by herpes virus encoded enzyme, thymidine kinase, and inhibits viral DNA polymerase.

Spectrum: Herpes viruses. HSV-1, HSV-2, VZV, and EBV.

Adverse effects: Nephrotoxicity, tremors, local irritation depending on route of administration.

GANCICLOVIR

Mechanism: Similar mechanism to acyclovir but with a preference for the inhibition of CMV DNA polymerase

Spectrum: CMV

Adverse effects: neutropenia, nephrotoxicity

HIV Therapy

REVERSE TRANSCRIPTASE INHIBITORS

Nucleosides: Zidovudine (AZT), Didanosine, Zalcitabine, Abacavir

Nonnucleosides: Nevirapine, Delaviridine, Efavirenz

Mechanism: Inhibits HIV reverse transcriptase and thus preventing the incorporation of HIV into host genome

Adverse effects: Bone marrow suppression, megaloblastic anemia, peripheral neuropathy, rash, and lactic acidosis

Protease Inhibitors: Ritonavir, Saquinavir, Nelfinavir, Amprenavir

Mechanism: Blocks protease enzyme thereby inhibiting assembly of new viruses

Adverse effects: Thrombocytopenia, GI irritation, hyperglycemia, and lipid abnormalities

► ENDOCRINE

Diabetes Medications

INSULIN

Mechanism: Binds to insulin receptors. In muscles, stimulates uptake of glucose, stimulates glycogen and protein synthesis, and stimulates K^+ uptake. In adipose tissues, stimulates uptake of glucose and stimulates triglyceride storage. In liver, stimulates increase storage of glucose as glycogen.

Clinical use: Diabetes and life-threatening hyperkalemia.

Toxicities: Hypoglycemia.

SULFONYLUREAS

Glipizide, Glyburide, Tolbutamide, Chlorpropamide

Mechanism: Stimulation of insulin release from beta-cells of pancreas; stimulates binding of insulin to target tissues; decreases serum glucagon levels

Clinical use: Type II diabetes (requires presence of beta-cells)

Toxicities: Hypoglycemia and disulfiram reactions (tolbutamide and chlorpropamide)

BIGUANIDES

Metformin

Mechanism: Decrease hepatic gluconeogenesis

Clinical use: Type II diabetes

Toxicities: Lactic acidosis

GLITAZONES

Rosiglitazone, Pioglitazone

Mechanism: Increases target cell response to insulin

Clinical use: Type II diabetes

Toxicities: hepatotoxic, weight gain

ALPHA-GLUCOSIDASE INHIBITOR

Acarbose, Miglitol

Mechanism: Inhibits alpha-glucosidase in intestinal brush border and therefore decreasing the absorption of starch and disaccharides

Clinical Use: Type II diabetes

Toxicities: Flatulence, diarrhea, abdominal cramping

Hyperthyroidism

PROPYLTHIOURACIL, METHIMAZOLE

Mechanism: Inhibition of the coupling and organification steps of thyroid hormone synthesis. Propylthiouracil also decreases the peripheral conversion of T_4 to T_3 .

Toxicities: Aplastic anemia, agranulocytosis, rash.

Steroid Therapy

GLUCOCORTICOIDS

Prednisone, Triamcinolone, Dexamethasone, Beclomethasone, and Hydrocortisone

Mechanism: Binds to specific intracellular cytoplasmic receptors in tissues. Promotes normal intermediary metabolism to provide increased resistance to stress. Decrease the production of leukotrienes and prostaglandins by inhibiting phospholipase A2 and expression of Cox-2.

Clinical uses: Inflammation, Addison's disease, immune suppression, and asthma.

Toxicities: Iatrogenic Cushing's syndrome.

Analgesics

OPIOIDS

Fentanyl, Morphine, Meperidine, Methadone, Sulfentanil, Codeine, Heroin, Dextromethorphan

Mechanism: Interacting with opioid receptors (μ , δ , κ) in the CNS and GI tract to modulate synaptic transmission.

Clinical uses: Pain, depression of cough reflex (dextromethorphan), diarrhea (loperamide).

Toxicities: Respiratory depression, constipation, miosis, additive CNS depression and addiction.

NSAIDs

Aspirin, Ibuprofen, Indomethacin, Naproxen, Etodolac

Mechanism: Aspirin irreversibly inhibits Cox-1 and Cox-2, while the other NSAIDs are reversible inhibitors of Cox-1 and Cox-2. Prostaglandin synthesis is also blocked.

Clinical uses: Analgesia, anti-inflammatory, and antipyretic.

Toxicities: Aplastic anemia, GI ulcers, GI irritation, and Reye's syndrome.

ACETAMINOPHEN

Mechanism: Inhibition of cyclooxygenase in CNS.

Clinical uses: Antipyretic and analgesic.

Toxicities: Hepatotoxicity with overdose. Acetaminophen metabolite depletes glutathione and forms toxic metabolite, NAPQI, in liver. *N*-acetylcysteine is antidote.

COX-2 INHIBITORS

Celecoxib, Valdecoxib

Mechanism: Preferential inhibition of COX-2 isoform that is found in inflammatory cells and mediates pain and inflammation. Spares COX-1, which is helpful in maintaining gastric mucosa cells.

Clinical uses: Rheumatoid and osteoarthritis.

Toxicities: Risk of MI and strokes.

Autacoids

PROSTAGLANDINS

Misoprostol, Carboprost, Dinoprost, Dinoprostone

Clinical uses: Abortions, induction of labor, prevention of peptic ulcers and maintenance of patent ductus arteriosus

Toxicities: Diarrhea

Antihistamines

H₁ BLOCKERS

Reversible H₁ histamine receptor blockers

1ST GENERATION

Diphenhydramine, Dimenhydrinate

Clinical uses: Allergy, motion sickness and nausea, and sedatives

2ND GENERATION

Fexofenadine, Loratidine

Clinical uses: Allergy, far less sedating than 1st generation

Toxicities: Sedation, antimuscarinic, antialpha adrenergic effects

H₂ BLOCKERS

Reversible H₂ histamine receptor blockers

Cimetidine, Ranitidine, Nizatidine, Famotidine

Clinical uses: Peptic ulcers, esophageal reflux, gastritis

Toxicities: Potent inhibitor of P-450 (cimetidine)

Prosthodontics

Jin Ha Joung, DMD

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► GENERAL CONSIDERATIONS

Prosthodontics is the dental specialty pertaining to the diagnosis, treatment planning, rehabilitation and maintenance of the oral function, comfort, appearance, and health of patients with clinical conditions associated with missing or deficient teeth and/or oral and maxillofacial tissues using biocompatible substitutes.

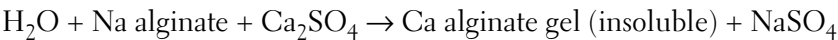
Diagnostic Procedures

IMPRESSIONS



The first reaction to take place is when sodium phosphate reacts with soluble calcium ions. Once the sodium phosphate is depleted, the actual setting reaction will begin.

Alginate (irreversible hydrocolloid): A hydrocolloid consisting of a solution of alginic acid having a physical state that is changed by an irreversible chemical reaction forming insoluble calcium alginate. The powder consists of different ingredients, each with their own properties (Table 3–1). The setting reaction is represented by the following formula:



Three ways to decrease setting time:

1. Increase water temperature
2. Mix more rapidly
3. Decrease water-powder ratio

After disinfection, casts should be poured immediately due to the following reasons.

- **Imbibition**, which is the process of absorbing water leads to alginate expansion.
- **Syneresis**, which is the exudation of the liquid component of a gel leads to alginate shrinkage.

TABLE 3 – 1. Alginate Powder Ingredients and Properties

INGREDIENT	DESCRIPTION	PERCENTAGE
Sodium alginate	Reactor, dissolves in water and reacts with calcium ions to form the insoluble gel	18%
Calcium sulfate dehydrate	Reactor, reacts with sodium alginate to form an insoluble calcium alginate gel	14%
Diatomaceous earth	Filler, controls consistency of mix and flexibility of set impression	56%
Potassium sulfate	Counteracts inhibiting effect of alginate on stone surface	10%
Sodium phosphate	Retarder, controls setting time (fast or slow)	2%



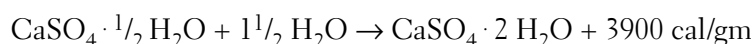
Permanent deformation is time dependent → quick “snap” removal will minimize distortion of alginate.

TABLE 3-2. Calcium Hemihydrate Cast Materials

TYPE	COMMON NAME	CRYSTAL STRUCTURE
α	dental stone	dense, prismatic shape
β	plaster of Paris	spongy, irregular shape

CASTS

- **Calcium sulfate hemihydrate:** This is the principal component of dental gypsum products, which are used for creating casts for diagnosis and dental prosthesis fabrication. There are two types of calcium hemihydrate obtained through two different methods of calcinations (Table 3-2). Dental stone is classified into five types based on their properties and applications (Table 3-3). The setting reaction is represented by the following formula:



Clinical Guidelines

- Water-powder ratio—follow manufacturer's instruction
 - $\downarrow$ water $\rightarrow$ $\downarrow$ setting time
- Water temperature
 - $\uparrow$ temperature $\rightarrow$ $\downarrow$ setting time
- Vacuum mixing—mixing below atmospheric pressure, which produces a smooth and bubble-free mixture
 - $\downarrow$ setting time, $\downarrow$ setting expansion, and $\uparrow$ compressive strength
- Vibrator—used when pouring stone into impression to eliminate trapped air bubbles

TABLE 3-3. Classification of Dental Stone

TYPE	ADA SPECIFICATION	TRADITIONAL NAME	APPLICATION
I	Impression plaster	Impression plaster	Impressions of flabby tissue for dentures, but rarely used
II	Model plaster	Lab plaster	Casts where strength is not important (ortho)
III	Dental stone	Class 1 stone	Diagnostic casts, casts for denture processing
IV	High-strength dental stone	Class 2 or improved stone	Master casts for die fabrication
V	High-strength and high-expansion dental stone		



Arcon vs. Nonarcon. An arcon articulator has the condylar elements in the lower member more closely resembling our jaws and joints in comparison to a nonarcon articulator which has the condylar elements in the upper member. This affects the arc of closure in that the angle between the condylar inclination and occlusal plane changes with a nonarcon articulator, while the angle remains the same on an arcon articulator.

ARTICULATORS

Articulator: A mechanical instrument, representing the temporomandibular joints and jaws, to which casts may be mounted to simulate some or all mandibular movements. They can be classified into four types:

Class	Name	Description
I	Nonadjustable	Accepts a single static registration; vertical motion is possible on some (used for functionally generated path technique)
II	Nonadjustable	Horizontal and vertical motion is possible, but the movements are not oriented to the temporomandibular joints (TMJs)
III	Semiadjustable	An instrument that simulates condylar pathways by using averages or mechanical equivalents for all or part of the motion; these instruments allow for orientation of the casts relative to the TMJs and may be arcon or nonarcon
IV	Fully adjustable	An instrument that will accept three-dimensional dynamic registrations; these instruments allow for orientation of the casts to the TMJs and simulation of mandibular movements

MAXILLOMANDIBULAR RELATIONS

Facebow is an instrument used to record the orientation of the maxillary arch to some anatomic reference point or points and transfer this information to the articulator; it allows the mounting of the maxillary cast to the upper member of the articulator.

Reference points vary depending on the facebow, and may include nasion or orbitale. These points are based on averages and yield an error of 2 mm or less in most patients.

- **Arbitrary vs. kinematic:** Arbitrary facebows use average anatomic landmarks to approximate the actual hinge axis. Otherwise, the actual hinge axis can be located using a kinematic facebow.

Interocclusal Records

- Bite registration—usually made with wax or an elastomeric material such as polyvinylsiloxane
- Centric relation record—allows one to mount the relationship of the mandibular cast to the maxillary cast in centric relation (CR)

- Protrusive record—allows one to set the condylar inclination on the articulator
- Lateral excursion records—allows one to set the Bennet angle on the articulator

Pantograph is an instrument used to graphically record in one or more planes, paths of mandibular movement in order to provide information for the programming of a fully adjustable articulator.

Functionally generated path (FGP): A registration of the paths of movement of the occlusal surfaces of one arch onto a recording medium attached to the opposing preparations. It allows the precise fabrication of the morphology of the occlusal surfaces of the teeth being restored.

Treatment Planning

ESTHETICS

The esthetics of a person can be evaluated in three ways. The overall facial evaluation consists of symmetry, midlines, and proportions. The dental evaluation comprises the shape, color, and positions of the teeth, while the soft tissue evaluation includes the gingival framework for the teeth. The ultimate objective is to create teeth that are in harmony with the gingiva, lips, and face.

- **Facial proportions:** The face from the frontal plane can be divided into thirds. The upper third is from the hairline to the brows. The middle third is from the brows to the bottom of the nose. The lower third is from the bottom of the nose to the bottom of the chin.

The lower third of the face can be further divided into thirds: the upper third and the lower two-thirds with the dividing line between the upper and lower lips.

- **Interpupillary line:** Serves as a horizontal reference for the orientation of the maxillary teeth and incisal/occlusal plane.

Smile Analysis

Dental midline ideally should coincide with the facial midline, but studies show that midlines up to 2 mm off center are not noticeable unless they become canted obliquely.

Incisal display at rest is age dependent and in part determined by the underlying skeletal structure, but the averages are 2 mm for males and 3.5 mm for females. As people become older, less of the maxillary incisors show at rest and more of the mandibular incisors can be seen.

Incisal and gingival display for a smile include the following ideal guidelines:

- Excessive gingival display (“gummy” smile) >3 mm of gingiva
- Gingival heights of the maxillary lateral incisors are 1 mm coronal to the heights of adjacent teeth
- Maxillary incisal edge position—slight convexity which follows the lower lip line
- Gull wing effect—the outline of the incisal edges from canine to canine looks like a gull’s wing due to the position of the lateral incisal edge being about 1 mm more apical than the canine cusp tips and the central incisal edge positions



Golden Proportion: 1.618:1

- Occlusal plane—posteriorly, the occlusal plane should follow a graduated curve of Spee from the canines to the molars
- Buccal corridors—the black space between the buccal aspects of the posterior teeth and the corners of the mouth when smiling

Golden proportion is the mathematical ratio denoted by the Greek letter phi (ϕ) which is claimed to explain natural beauty. In dentistry, some say that the golden proportion exists in esthetic natural dentitions when viewing the widths of the maxillary incisors and canines from the direct facial view.

Dental Esthetics

Three overall tooth shapes: ovoid, square, and tapering

PHONETICS

Rothman's list of essential mechanisms for speech production:

- **Initiator**—Motor speech area of the brain and the nerve pathways
- **Motor**—Lungs and associated musculature
- **Vibrator**—Vocal cords
- **Resonator**—Oral, nasal, and pharyngeal cavities and paranasal sinuses
- **Enunciators/articulators**—Lips, tongue, soft palate, hard palate, and teeth

Speech sounds (anatomic sound formation):

- **Linguopalatal** (or palatolingual)—sh (shoe), z (vision), ch (chin), j (jar), r (rose), y (you)—made with the palate
- **Linguovelar**—k, ng, g—made with the posterior dorsal tongue raised to occlude with the soft palate
- **Linguoalveolar**—t, d, n, l, s—made with the valve formed by contact of the tip of the tongue with the most anterior part of the palate (the alveolus) or the lingual side of the anterior teeth
- **Linguodental**—th—made with the tip of the tongue extending slightly between the maxillary and mandibular incisal edges (about 3 mm)
- **Labiodental**—f, v—made between the upper incisors and the labiolingual center to the posterior third of the lower lip (wet-dry junction)
- **Bilabial**—b, p, m—made by contact of the lips

Closest speaking space is the distance between the incisal edges of the mandibular incisors to the palatal surfaces of the maxillary incisors when producing the /s/ sound (e.g., sixty-six or Mississippi). It gives the vertical dimension of speech. Important for evaluating vertical dimension.

Maxillary incisal edge position relative to the wet-dry junction of the lower lip produces the /f/ or /v/ sound (e.g., fifty-five).

The /m/ sound (e.g., Emma) is useful in bringing the mandible to its rest position.

OCCCLUSION

Mandibular teeth act as a **class III lever**. An example is a fishing pole. Class III levers generate 4 the work in the molar region.

- **Temporomandibular joint (TMJ)**: The TMJ is a ginglymodiarthrodial joint, which allows both rotation and translation of the mandible.

- **Posselt's envelope of motion:** Describes border movements in both the sagittal and frontal planes.

Four determinants of **mandibular movement**:

1. Anterior guidance
2. Right condylar assembly
3. Left condylar assembly
4. Neuromuscular control

General Terms

- **Vertical dimension of occlusion (VDO) or occlusal vertical dimension (OVD):** The vertical dimension of the face when the teeth are in centric occlusion.
- **Vertical dimension of rest (VDR) or physiologic rest position:** The vertical dimension of the face when the mandible is in the rest position.
- **Freeway space (FS):** The space between the maxillary and mandibular teeth when the mandible is at rest, sometimes called interocclusal distance.
- **Centric relation (CR):** The most confusing term in prosthodontics. Even the Glossary of Prosthodontic Terms 8th edition lists seven different definitions. Basically, CR is the position of the mandible (or specifically the location of the condyles in their respective glenoid fossae) from which pure rotary movement can occur independent of the teeth. Most importantly, it is theoretically a repeatable position to which a patient can be restored.
- **Centric occlusion (CO):** The occlusion of opposing teeth when the mandible is in centric relation.
- **Maximum intercuspal position (MIP):** The occlusion of opposing teeth when they are in maximum intercuspation.
- **Rest (postural) position:** The position of the mandible at physiologic rest.

Bennett movement is an obsolete term. The correct term is laterotrusion, which is movement of the working side condyle in the horizontal plane.

Immediate mandibular lateral translation (immediate side shift) is movement of the nonworking side condyle in the horizontal plane immediately as it leaves from CR in a straight and medial direction (or the working side condyle in a lateral direction).

Occlusal Schemes

- Mutually protected occlusion (natural teeth)
 - MIP: Primary occlusal axially directed loading is absorbed by supporting cusps on posterior teeth; anterior teeth contact very lightly and should not bear heavy forces
 - Protrusion: Overbite–overjet relationship of the incisors produces an incisal guidance that causes disclusion of all posterior teeth
 - Lateral excursion: Overbite–overjet relationship of contacting teeth on working side should cause disclusion of all nonworking side teeth
 - Canine guidance: Working side canines disclude all other teeth
 - Group function: Working side canines, premolars, and molars disclude all nonworking side teeth
- Balanced articulation (dentures)
 - MIP: Some believe only the posterior teeth should contact, whereas others believe the anterior teeth should contact as well
 - Protrusion: Anterior and posterior teeth contact
 - Lateral excursion: Equal working and nonworking side contacts



$$VDO = VDR - FS$$



$$\text{Freeway space} = 1-3 \text{ mm}$$



$$\begin{aligned} \text{Maximum opening} = \\ 40-50 \text{ mm (roughly about} \\ \text{four finger widths)} \end{aligned}$$



Typical abfraction lesion is a V-shaped notch at the cervical margin.



Ferrule = 1.5–2.0 mm of vertical tooth structure above the finish line



Epulis Fissuratum—overgrowth of intraoral tissue caused by chronic irritation, usually an overextended denture flange.



Kelly (Combination) syndrome = specific changes caused by a mandibular Kennedy class I removable partial denture opposing a maxillary complete denture.



1-stage vs. 2-stage refers to the number of surgeries needed before restoring the implant.

Tooth Wear

- **Attrition:** Normal wear of occlusal and/or incisal surfaces of opposing teeth during mastication, but can turn excessive with parafunction
- **Abrasion:** Abnormal wear due to a mechanical process other than mastication (e.g., toothbrush)
- **Erosion:** Wear due to chemical means (e.g., bulimia, GERD)
- **Abfraction:** Still controversial with questionable etiology, but the currently accepted cause is biomechanical loading forces leading to flexure fatigue degradation at a distant location on the tooth

Preprosthodontic Treatment

Prosthetic phase of treatment is the last stage of rehabilitation. Before the prosthodontic phase is started or during the course of treatment, other phases of treatment may be needed, including but not limited to endodontic, orthodontic, and/or periodontal treatment.

- **Crown lengthening:** Teeth without adequate tooth structure (to develop a ferrule) may need surgical crown lengthening to remove osseous structure to expose more tooth structure.
- **Forced eruption or orthodontic extrusion** is another option for teeth without adequate tooth structure, but better than crown lengthening surgery because it results in a better crown-to-root ratio and better esthetics.
- **Ridge augmentation:** Soft and/or hard tissue augmentation of edentulous ridges or pontic spaces.
- **Removal of flabby tissue:** Excess or too much soft tissue provides poor support for a denture and in certain instances can be surgically reduced to provide a better area for support.
- **Inflammatory papillary hyperplasia (IPH)**—hyperplasia of palatal tissue under a poor fitting denture base that is kept in at night.

TORI REMOVAL

- Maxillary tori—usually located in the middle of the hard palate; depending on the size and location, can be left alone
- Mandibular tori—almost always has to be removed for denture fabrication

Tuberosity reduction: Enlarged tuberosities can interfere with placement of denture teeth in proper occlusal plane, especially with Kelly syndrome (also called combination syndrome).

Vestibuloplasty can be performed to increase the depth of the vestibule using skin grafts.

Implant Prosthodontics

- **Osseointegration:** Brånemark ushered in the modern era of implantology with his breakthrough research of titanium root form implants. Osseointegration is the direct attachment of bone to the surface of the implant (i.e., ankylotic connection).
- **1-stage vs. 2-stage:** Implants originated with a 2-stage surgical approach. The first stage is the surgical placement of the implant, which is covered by the soft tissue and allowed to heal undisturbed. A second surgical procedure is needed to uncover the implant and a healing abutment (transmucosal

component) is placed to allow the soft tissue to heal around it. Many implants are now placed in a single-stage approach, eliminating the need for a second surgery. The implant already has a transmucosal component built into the implant or the healing abutment is placed during the surgical placement of the implant.

- **Implant design:** Many designs have been tried, including press-fit cylinders, hollow baskets, screw type, and plates. The majority of implants today are a self-tapping screw type design. Thread designs vary as well. Most implants come in different diameters to accommodate different areas of the mouth.
- **Implant surface:** Implants started with a smooth machined surface. Modifications have been made to roughen the surface and promote osseointegration (improve bone to implant contact). The addition of hydroxyapatite (HA) to the surface was popular for some time, but fell out of favor due to the degradation of the HA to implant bond. Novel techniques are being developed to promote faster osseointegration, including the addition of fluoride to the surface and the use of an oxygen-free manufacturing process.
- **Abutment:** The component which attaches to the implant and supports or retains the prosthesis. Abutments can be prefabricated or custom made and can be made of different materials, including titanium, alloys, and ceramics.
- **External vs. internal connection:** The connection between the abutment and the implant is a critical factor. The original Brånemark implant had an external hex on top of the implant to allow it to be driven into the bone. However, mechanical problems including screw loosening and fractures led to the development of internal connections. Another kind of internal connection used by some implant manufacturers is the Morse taper design, which is a mechanically locking friction fit connection.
- **Screw-retained vs. cement-retained:** Fixed prostheses can be either cemented to the abutment or retained through occlusal or transverse screws depending on the clinical situation.



The UCLA abutment is synonymous with a custom abutment.

► REMOVABLE PROSTHODONTICS

Complete Dentures

DIAGNOSIS AND TREATMENT PLANNING

Diagnosis of denture problems:

- Burning sensation in mandibular ridge → pressure on mental foramen
- Burning sensation in palatal area → pressure on incisive foramen
- Clicking of denture teeth → excessive VDO, porcelain teeth
- Cheek biting → not enough horizontal overlap of posterior teeth, insufficient OVD
- Decreased salivary flow → protein deficiency
- Angular cheilosis, glossitis, edema, and papillary atrophy → vitamin B deficiency, insufficient OVD
- Mucosal changes → vitamin C deficiency

Personality assessment made using **House's psychological classification**

- Philosophical—accepts dentist's judgment and instructions, best prognosis
- Exacting—methodical and demanding, asks a lot of questions, good prognosis
- Indifferent—doesn't care about dental treatment and gives up easily
- Hysterical—emotionally unfit to wear dentures, never happy, worst prognosis

Hard palate shapes flat, round, U, or V

House's palatal throat form—based on imaginary line drawn between hamular notches

- Class I—5–13 mm posterior, provides the most amount of tissue for post dam → ideal
- Class II—3–5 mm posterior, usually enough tissue for post dam → favorable
- Class III—3–5 mm anterior, not enough tissue for post dam → poor

Neil's lateral throat form—amount of space available to extend the lingual flange of the mandibular denture

- Class I—large → favorable
- Class II—in between
- Class III—small → unfavorable

Wright's tongue position

- Class I—normal → favorable
- Class II—retracted → unfavorable

Knowing the anatomy of the edentulous ridges and the anatomical features that determine the borders of the dentures are critical in fabricating successful complete dentures.

Direction of ridge resorption:

- **Maxilla**—superior and posterior
- **Mandible**—inferior and anterior

Posterior palatal seal (or post dam) is the posterior border of the maxillary complete denture that puts pressure on the displaceable tissue near the junction of the hard and soft palates aiding in retention of the prosthesis. Also compensates for the shrinkage of the acrylic resin during processing of the denture base.

Vibrating line is an imaginary line demarcating the moveable and nonmoveable tissues of the soft palate under function, which is used to determine the posterior extent of the maxillary complete denture.

Abused tissues need to be addressed before fabricating new dentures.

- Educate the patient
- Removal of unacceptable dentures from the mouth for an extended period of time before impressions
- Reline the dentures with a tissue conditioner
- Massage of the tissues and warm saline rinses

Three critical factors for successful complete dentures:

1. Stability—resistance of the denture base against lateral forces
2. Support—resistance to the forces directed against the tissues
3. Retention—resistance to dislodgement of the denture base away from the tissues

Immediate dentures—a complete denture that is delivered immediately following the extraction of any remaining teeth. Due to the resorption and remodeling that occurs after extraction, the dentures will need relines or be remade after minimum of 6 months.

Overdentures—a complete denture that is partially retained, supported, and/or stabilized with the help of teeth or implants. Different types of attachments can be used for overdentures including bars and stud-type attachments.

IMPRESSIONS

Diagnostic impressions are made with alginate using metal edentulous trays. Custom trays are fabricated on the diagnostic casts.

Final impression technique—different philosophies have existed over the years for complete denture final impressions

- Pressure/functional—tissues are compressed during impression to simulate the same amount of pressure during function, that is, mastication
- Nonpressure/mucostatic—tissues are impressed at rest without any pressure
- Selective pressure—pressure is selectively applied to those areas best suited for withstanding the forces of mastication using a custom tray which provides more relief over nonstress bearing areas

Polysulfide (rubber base) is an elastomeric impression material that has an exothermic setting reaction with water as a by-product (see Table 3–4)

- Advantages:
 - long working time
 - flexible and tear resistant
- Disadvantages:
 - long setting time
 - very unpleasant odor and taste
 - highest permanent deformation

Border molding is the shaping of the denture border areas with impression material typically compound. The shape and size of the vestibule is duplicated by either functional and/or manual manipulation of the soft tissue adjacent to the borders.

Modeling compound:

- Comes in stick or cake form
- Green melts at 123°F, red melts at 132°F



Polysulfide impressions must be poured within 30 minutes.

TABLE 3–4. Polysulfide Components

BASE	CATALYST
Mercaptan rubber containing sulfhydryl groups (-SH)	Lead dioxide or <i>tert</i> -butyl-hydroperoxide
80% low-molecular-weight polymer	Dibutyl phthalate
20% reinforcing agents that modify viscosity and increase strength	Sulfur



*Bead of adequate
width = 3/8"*

*Cast thickness = 16 mm from
the highest spot on the
impression.*

Beading and boxing—final impressions are beaded with rope wax and boxed with boxing wax to create master casts with proper land areas along the borders of the impression and an adequate base.

Record base and occlusal rim—an interim denture base is fabricated on the master cast to support the occlusal rim. The rim is made of wax to help with jaw relation records and the setting of teeth. The rim is contoured and adjusted in the mouth.

Maxillomandibular Relations

- Facebow transfer
- Determination of VDO
- CR record

Occlusion

SELECTION OF TEETH

Materials: acrylic resin or porcelain—almost all denture teeth today are made of plastic

Anterior tooth selection is based on 3 S's: shape, size, and shade

- Three basic shapes: rectangular/square, tapering/triangular, and ovoid or combinations
- It is determined by the mesial-distal distance available based on the contoured wax occlusal rim
- Shade is a subjective selection determined by the patient
- Arrangement of the anterior teeth:
 - Follow rules of esthetics and phonetics
 - Characterization based on the concept of dentogenics introduced by Frush and Fisher, which says the set-up should harmonize with the patient's sex, personality, and age

Posterior tooth selection is based on the occlusal scheme that is selected for that patient.

- Tooth form is based on cusp angle
- anatomic: 33–45 degrees → balanced occlusion
- semi-anatomic: 10–20 degrees → balanced occlusion
- nonanatomic/flat: 0 degree → monoplane occlusion (lingualized occlusion can use different combinations)

OCCUSAL SCHEMES

Balanced occlusion—working and nonworking side contacts of all posterior teeth during lateral excursive movements, posterior and anterior contacts in protrusion

- Advantages:
 - better food penetration due to the steeper cusps
 - more esthetic
- Disadvantages:
 - technically challenging to execute
 - steeper cuspal inclines can possibly lead to harmful lateral forces on the ridges

Lingualized occlusion—generally a balanced type of occlusion that puts emphasis on the maxillary lingual cusps occluding against flatter mandibular teeth

- Advantages:
 - still a balanced occlusion, but allows more flexibility
- Disadvantages:
 - less efficient mastication than balanced occlusion
 - more wear

Monoplane occlusion—posterior teeth are set either flat to flat or with a compensating curve to achieve balance; used especially for patients with a skeletal class II or class III profile

- Advantages:
 - More adaptable for difficult jaw relations
 - Can be used in cross-bite
- Disadvantages:
 - Poor penetrating power due to lack of cusps
 - Less efficient mastication
 - Poor esthetics

Hanua's quint gives the five principal factors or laws of articulation that govern balanced occlusion:

1. Condylar guidance
2. Incisal guidance
3. Compensating curve (combination of the curves of Wilson and Spee)
4. Relative cusp height
5. Plane of orientation

Thielemann turned Hanua's Quint into a formula for balanced occlusion:

$$K = (CG * IG) / (CC * CH * PO)$$



REALEFF—resiliency and like effect—resiliency of the soft tissues compensates for the limitations of the articulator.

DENTAL MATERIALS

Acrylic resin or polymethyl methacrylate (PMMA)—the material of choice for dentures (see Table 3–5)

TABLE 3–5. Acrylic Resin Components

POWDER	LIQUID (MONOMER)
Polymer beads	Methylmethacrylate
Benzoyl peroxide (initiator)	Hydroquinone (inhibits polymerization during storage)
Pigments, dyes, opacifiers, organic fibers	Plasticizers



BULL Rule for selective grinding—**Buccal Upper**,
Lower Lingual.

Activator for PMMA

- Auto-polymerized—tertiary amine or dimethyl-p-toluidine (monomer)
- Heat-polymerized—heat

PMMA undergoes shrinkage during polymerization

- Auto-polymerized—0.2%
- Heat-polymerized—3–7%

Processing—there are many ways to process the dentures, but the conventional technique involves a stone investment in a metal flask and boil out procedure, followed by a curing cycle with heat under compression.

- Dentures are invested in a flask
- The wax is boiled out from the investment
- Acrylic resin is packed and heat processed

Processing errors:

- porosity

Lab remount—after processing, but before the dentures are removed from the cast, they are remounted on the articulator to adjust the changes in occlusion that usually occur due to processing.

INSERTION AND POSTINSERTION

Delivery Steps

1. Fit—each denture is individually tried in the mouth for proper fit using a pressure indicating paste and adjusted for any pressure areas, and the orders are verified for proper extensions
2. Clinical remount—allows for precise adjustment of occlusion on the articulator
 - a. Remount casts are made in advance
 - b. Facebow transfer is preserved with a jig
 - c. New CR record is taken using the final dentures and mounted on the articulator
3. Occlusion—final adjustments made intraorally
4. Final polish
5. Patient instructions

Post-insertion visits

- 24 hours
- 1 week
- 1-year recalls: to check for fit of dentures and the need for a reline or even replacement

Reline vs. rebase

- Rebasings is the replacement of the entire denture base while keeping the same denture teeth in their current occlusal relationship
- Relining is the replacement of the intaglio surface of the denture base with a new layer of material
 - Reline materials can be soft (temporary) or hard (permanent)

Removable Partial Dentures

DIAGNOSIS AND TREATMENT PLANNING

- Goals of treatment:
 - Elimination of all disease
 - Restoration of function and esthetics
 - Preservation of remaining hard and soft tissues
- Considerations for RPD treatment:
 - Healthy ridges
 - Healthy abutment teeth
 - Interarch relationships
- Indications for RPD treatment:
 - Long-span edentulous areas
 - No posterior abutment tooth
 - Reduced periodontal support
 - Cross-arch stabilization
 - Excessive bone loss within the residual ridge
 - Patient desires
 - Physically or emotionally handicapped
 - Young age
 - Unfavorable maxillomandibular relationships
- Kennedy classification—partially edentulous arches are classified according to the most posterior edentulous area.
 - Class I—bilateral distal extension
 - Class II—unilateral distal extension
 - Class III—all tooth supported
 - Class IV—single anterior edentulous area crossing the midline
- Applegate's rules for applying the Kennedy classification
 - the classification should follow, not precede extractions
 - if a third molar is missing and not to be replaced, it's not considered in the classification
 - if a third molar is present and not to be used as an abutment, it's not considered in the classification
 - if a second molar is missing and not to be replaced, it's not considered in the classification
 - the most posterior area always determines the classification
 - edentulous areas other than those determining the classification are referred to as modifications and are designated by their number
 - the extent of the modification is not considered, only the number of additional edentulous areas
 - there are no modification areas in a class IV

All diagnostic procedures must be started with mounted diagnostic casts and a preliminary survey and design.

RPD DESIGN

RPD design starts with a survey of the casts and depends on the specific anatomic features of the arch and the remaining dentition.



A Kennedy Class IV RPD cannot have modification spaces.



Surveying is the process of locating the heights of contour on abutment teeth to help in designing the RPD.

Important design considerations for tooth-borne versus tooth- and tissue-borne RPDs:

- significant difference exists between the support that teeth can provide versus the support of the residual ridge
 - healthy teeth can move up to 0.2 mm under function due to the periodontal ligament
 - soft tissue overlying the bone can be displaced 1.0 mm or more
- understanding of the mechanics of how a RPD will move under function is critical in designing a successful RPD
 - multiple fulcrum lines can exist from one abutment tooth to another
- Class I RPDs require special attention in their design and need to incorporate:
 - maximum support of the distal extension bases
 - flexible direct retention to prevent torque of the abutment teeth
 - indirect retention to prevent lifting of the denture bases away from the tissues
- Class III RPDs have less demands than the class I due to it being completely supported by the teeth
 - less movement, almost as good as an FPD
 - Indirect retention is NOT necessary
- Class II RPDs need to feature elements from both class I and II RPD design characteristics
- Class IV RPDs can act in several ways depending on the length of the edentulous span

Components of a RPD:

- Major connectors
- Minor connectors
- Rests
- Direct retainers
- Indirect retainers
- Denture bases
- Denture teeth



Major connector = RIGIDITY



8 mm is necessary for a lingual bar (3 mm away from gingival margins + 5 mm for the bar).

Major connector—connects the components of the two sides of the arch together

- Maxillary major connectors:
 - Posterior palatal strap
 - U-shaped or horseshoe
 - Anterior-posterior (A-P) palatal strap
 - Full palatal plate
- Mandibular major connectors:
 - Lingual bar—most common
 - Lingual plate
 - Kennedy bar (double lingual bar)
 - Labial bar

Minor connectors connect all the remaining components of the RPD to the major connector and provide stress distribution.

- Components include clasp assemblies, direct retainers, indirect retainers, auxiliary rests, and denture bases

Rests and Rest Seats

- Rests prevent displacement of the RPD toward the tissue and transfers the forces of mastication to the supporting teeth

- Rests are placed as part of a retentive clasp assembly
- All teeth adjacent to edentulous spaces need a rest
- Rest seats are prepared into teeth with the following guidelines:
 - Occlusal rests:
 - Triangular in shape with the base located at the marginal ridge
 - $1/3 - 1/2$ the mesial-distal width
 - $1/2$ the buccolingual width from cusp tip to cusp tip
 - Floor of the rest preparation must be inclined toward the center of the tooth
 - Thickness of the metal should be 1.0–1.5 mm
 - Cingulum (lingual) rests:
 - Usually used on maxillary canines
 - V-shaped in cross section
 - Crescent shaped when viewed from the lingual
 - Incisal rests:
 - Usually used on mandibular canines
 - Small V-shaped rounded notch located 1.5–2.0 mm away from the proximal-incisal angle
 - Extended slightly onto the facial surface

Direct retainers prevent the RPD from moving away from the hard and soft tissues.

Classification of direct retainers:

- Intracoronal (located within the contours of the abutment crown):
 - Precision attachments
 - Semiprecision attachments
- Extracoronal (located outside the contours of the abutment crown):
 - Retentive clasp assemblies:
 - Infrabulge
 - Suprabulge
 - Attachments

Extracoronal attachments come in many different configurations. One popular example is an ERA. These attachments are generally incorporated into the wax pattern for the crown of the abutment tooth and cast together in metal.

Retentive Clasp Assemblies

- Four components include the retentive arm, reciprocal arm, rest, and guiding plate
- Terminal end of the retentive clasp engages an undercut; it is the only part of the assembly below the height of contour on the tooth
- Clasp requirements:
 - As little retention as needed
 - Reciprocation or bracing of retentive arm either with another clasp arm or minor connector
 - At least 180° of encirclement
 - Support with proper rests
 - Passive fit when fully seated; retentive portion of clasp only engages tooth when dislodging forces are applied (prevent orthodontic forces that might move the tooth)
- Types of retentive clasps base on how the clasp approaches the undercut:
 - Suprabulge—Aker's or circumferential (C-clasp)
 - Infrabulge—Roach or bar (I, J, T, L, etc.)



Cross section of lingual bar = half-pear shaped



Precision attachments are manufactured in metal, whereas semiprecision attachments are made of plastic or wax and cast in metal.



*Infra- = from below
Supra- = from above*



Indirect retainers are needed only for distal extension RPDs.

Clasp Design Principles

- Resiliency or flexibility of a clasp is determined by its length, diameter, taper, shape, and type of metal
- Type of metal:
 - Wrought wire—0.020 in. undercut
 - more flexible than cast metal
 - can be soldered, incorporated into the wax pattern before casting, or embedded in acrylic resin during processing
 - Cast metal—0.010 in. undercut
- Shape in cross section can be round or a half circle
- As taper increases, flexibility increases
- As diameter increases, flexibility decreases in a cube ratio
- As length increases, flexibility increases in a cube ratio
- The more the clasp is bent, flexibility decreases

RPI Design

- Stress-breaking design principles are used for distal abutments in a Kennedy class I or II RPD to prevent any pumping or extrusive forces that might be applied to the tooth when under function
- RPI stands for mesial **R**est and minor connector, distal guide **P**late, and **I**-bar, which engages a mesial undercut
- These three areas of contact with the teeth provide the required 180° encirclement
- Under function, the terminal end of the I-bar will move apically and mesially away from the tooth
- RPA is a modification of the RPI design by replacing the I-bar with an Aker's clasp

Indirect retainers prevent the distal extension from moving away from the underlying tissue during function

- Ideal location is determined by an imaginary line drawn perpendicular to the fulcrum line and as anterior as possible

Tissue stops provide a positive stop of the framework on the cast to prevent movement during processing

Finish lines are those interfaces where resin meets metal to provide a smooth transition

- External finish line $<90^\circ$
- Internal finish line $= 90^\circ$

MOUTH PREPARATION

Before mouth preparation can commence, all other procedures must be completed to return the mouth to a state of health, including:

- extraction of hopeless teeth and roots
- periodontal and/or endodontic treatment
- operative
- surveyed crowns to restore broken down teeth to ideal contours

Modification sequence:

1. parallel guiding planes
2. heights of contour
3. retentive contours
4. rests

Occlusion

Occlusion principles for RPDs involve a combination of complete denture occlusion and natural teeth occlusion. The type of occlusion depends on the opposing arches and the types of prostheses that are used. The general rule is that the prosthesis with the least amount of support determines the occlusal scheme. If either arch is a complete denture, then balance is recommended. If anterior guidance can be established with the remaining teeth, then anterior guidance is recommended.

DENTAL MATERIALS

- RPD metal framework materials:
 - Gold alloy—not used anymore
 - Base metal alloys:
 - Cobalt-Chromium (Co-Cr)
 - Nickel-Chromium (Ni-Cr)
- RPD bases are made of PMMA, just like complete dentures
- RPD teeth are the same as for complete dentures

INSERTION AND POSTINSERTION

RPD frameworks can be tried in the mouth and adjusted before processing.

Altered cast technique allows you to take a better or a functional impression of the edentulous areas in a distal extension type RPD using the framework like an impression tray.

RPDs are inserted and adjusted one at a time and then in combination. Techniques are similar to complete dentures involving the fit and occlusion.

Patients are usually instructed to remove the prosthesis at night, but some advocate keeping them in at night to prevent any movement of the teeth.

Recall is especially important for patients with RPDs to maintain healthy abutment teeth and tissues. The patient with a Kennedy class I mandibular RPD opposing a maxillary complete denture must be monitored to assess the fit of the RPD and the need for relines to prevent the Kelly syndrome.

Characteristic features of the Kelly (combination) syndrome:

- Bone loss in the anterior maxilla
- Overgrowth of maxillary tuberosities
- Papillary hyperplasia of the hard palate
- Supraeruption of the mandibular anterior teeth
- Bone loss underneath the distal extensions of the mandibular RPD

► FIXED PROSTHODONTICS

Design of Prosthesis and Mouth Preparation

TYPES OF SINGLE-UNIT INDIRECT RESTORATIONS

- Inlays
- Onlays
- 1/2 crown

- 3/4 crown
- 7/8 crown
- Full-metal crown
- Metal–ceramic crown (PFM—porcelain fused to metal)
- All ceramic crown
- Veneer

TYPES OF MULTIUNIT INDIRECT RESTORATIONS

- Maryland bridge (resin bonded fixed partial denture)
- Full-metal fixed partial denture
- Metal–ceramic fixed partial denture
- All ceramic fixed partial denture

Ante's law—not really a scientific law, but a recommendation that the surface area of the roots of the abutment teeth should equal or exceed the surface area of the roots of the teeth to be replaced.

Biologic width consists of the junctional epithelium and the connective tissue attachment to the tooth above the alveolar crest. When preparing teeth for full-coverage restorations, the biologic width and the sulcus must be accounted for when placing your margins. Encroachment of the biologic width can lead to chronic gingival irritation and inflammation or bone loss.

Crown-to-root ratio:

- Minimum—1/1
- Best—1/2

Tooth preparation starts with knowing the contours of the finished product based on a diagnostic wax-up and reducing enough tooth structure to accommodate the planned restoration.

- Biologic considerations:
 - Prevent pulpal injury
 - Maintain periodontal health
 - Conserve tooth structure
- Mechanical considerations:
 - Retention form—features of the preparation that resists dislodgment of the crown in a vertical direction or along the path of insertion
 - Resistance form—features of the preparation that allows the crown to resist lateral forces or any path other than the path of insertion

Tooth preparation features:

- Taper (total occlusal convergence)—ideal is 3–6 degrees, the more parallel the walls of the preparation, the better retention and resistance form
- Diameter of base
- Height—the taller the preparation, the better the resistance form
- Relationship of height-to-base ratio and taper is important in determining adequate resistance form
- Surface area and roughness—combination of preparation height and diameter, increase in surface area or roughness will increase retention form
- Boxes and/or grooves—helps improve resistance form, but can also provide some retention form

Finish line is dictated by the type of restoration that is planned.

- Chamfer—best for metal margins
- Shoulder—best for porcelain margins



$$BW = 1 \text{ mm (CT)} + 1 \text{ mm (JE)} = 2 \text{ mm}$$



Resistance form is more important than retention form.

- Featheredge or knife edge
 - Usually contraindicated due to overcountouring of the crown near the margin

Bevels are rarely indicated except for full-gold crowns.

- Margin design:
 - Porcelain margin (sometimes called a butt joint)—most esthetic
 - Metal margin—least esthetic
 - Disappearing metal margin—compromise between the two, but tends to become overcontoured and the porcelain fractures because it is placed under tension

Margin location can be supragingival, at the gingival crest, or subgingival

- Most hygienic is supragingival
- Indications for subgingival margins include:
 - Esthetics
 - Subgingival caries
 - Preexisting subgingival restoration
 - Root sensitivity
 - Improve resistance form

Ferrule is a term used to describe the circumferential remaining tooth structure above the margin. Minimum amount of sound tooth structure necessary is 1.5–2.0 mm, or the risk of failure increases.

- **Foundation restorations:** Many teeth in need of fixed prosthodontics require foundation restorations to replace missing tooth structure to provide adequate resistance and retention forms. Endodontically treated teeth can be a challenge to restore properly.

Post and Core Principles

- Some teeth can be restored with just a core material if it can be adequately retained by the remaining tooth structure.
- Pins can help retain cores in some situations.
- Many molars can be adequately treated with an amalgam dowel and core due to their large pulp chambers.
- A post is indicated if there is significant loss of tooth structure and the core cannot be adequately retained or the core will contribute to the retention and resistance forms.
- Post is placed into the largest root
 - Maxillary premolars/molars: palatal root
 - Mandibular molars: distal root
- Posts come in many different shapes and styles; they are either prefabricated or custom made as a metal cast post and core.
- Post length— $\frac{2}{3}$ to $\frac{3}{4}$ the length of the root in the bone or equal to the length of the clinical crown.
- Post width—critical to preserve as much radicular dentin around the post, but no guidelines exist.
- 4–5 mm of gutta percha should remain to seal the apical portion and provide resistance to microleakage.

Core materials: amalgam, composite resin, glass ionomer, resin-modified glass ionomer, cast metal



4–5 mm of gutta percha must remain for adequate seal



*Posts do not reinforce teeth.
There only purpose is to retain
the core.*

Custom Cast Post and Core

- Indicated for a severely mutilated tooth
- Antirotation feature can be placed if necessary
- Positive seat required to prevent over seating and wedging effect
- Typically cast using gold alloys
- Two ways to fabricate a pattern:
 - Direct pattern—best method, but requires more chair time
 - Indirect/impression—difficult to get accurate impression of canal space

Types of prefabricated posts:

- Materials: stainless steel, titanium, resin, zirconia, cast metal
- Parallel, tapered, or tiered
 - Tapered posts can have a wedging effect
 - Parallel posts require more apical dentin to be removed
- Passive or actively engage tooth structure
 - Posts with threads engage the dentin providing more retention, but generate unfavorable stresses that may lead to fractures (contraindicated)
- Rough (serrated) or smooth
- **Tissue retraction:** Accurate impressions start with adequate retraction of the gingival tissues away from the finish line in both a horizontal and vertical dimension. The goal is to provide space for enough impression material without tearing.

Retraction Cords

- Plain, braided, or knitted
- Different sizes and diameters
- Some cords come impregnated with a hemostatic agent such as epinephrine

Hemostatic agents—help control moisture (blood and saliva) around the finish line which would be detrimental to making an accurate impression.

- Epinephrine 0.1, 0.8%:
 - causes vasoconstriction
 - contraindicated for cardiac patients
- Potassium aluminum sulfate:
 - slightly less effective than epinephrine
 - 0.1 mm loss of tissue
- Aluminum chloride 5–10%:
 - if greater than 10%, will cause tissue destruction
 - 0.1 mm loss of tissue
- Ferric sulfate 13.3%:
 - good hemostatic agent
 - discolors tissue temporarily, so use with caution in the anterior
- Zinc chloride 8%, 40%:
 - causes tissue necrosis
 - no longer used
- Electrosurgery:
 - cauterization of tissues around finish line

Impression Procedures

Impression trays:

- Custom trays—made from self-curing acrylic resin or light-cured material
- Stock trays—plastic or metal

- Triple trays—simultaneously provides impression of the preparation, the bite registration, and the impression of the opposing arch

Many techniques for impression procedures are available, but the most common technique will be described.

Double-cord technique involves the use of a first cord placed into the sulcus which will remain during the impression procedure with a larger second cord placed and removed just before injecting impression material into the sulcus.

A wash material is injected around the preparations and a more rigid or putty material is placed into the tray.

Framework Design

- Most important design consideration for metal–ceramic prostheses is designing the framework to support an even thickness of porcelain (1–2 mm) throughout
- Minimum thickness of metal to support porcelain is 0.3 mm
- Opposing tooth contacts (reduction required):
 - Metal: 1.5 mm for functional cusp, 1.0 mm for nonfunctional cusp
 - Porcelain: 2.0 mm for functional cusp, 1.5 mm for nonfunctional cusp
 - Porcelain to metal junction should be at least 1.5 mm away from tooth contacts
- Connectors for fixed partial dentures:
 - Width:
 - halving the width → strength is reduced by 1/2
 - doubling the width → strength is doubled
 - Height:
 - halving the height → strength is reduced by 1/8
 - doubling the height → strength is cubed

Pontics are used to replace missing teeth as part of fixed partial dentures.

- Ridge lap:
 - Esthetic, but due to its concavity, hygiene is impossible resulting in inflammation → contraindicated
- Sanitary (hygienic):
 - Clearance of 2–4 mm from the ridge allowing patient to easily clean underneath, but space can become a food trap and annoyance to the tongue
 - Poor esthetics
- Bullet:
 - Similar to sanitary, but shaped like a bullet with the tip sitting against the ridge
 - Poor esthetics
- Modified ridge lap (Stein):
 - Modification of the ridge lap pontic, almost like a ridge lap on the buccal and a bullet on the lingual
 - Convex surface with pinpoint contact against the buccal aspect of the ridge
- Ovate:
 - Most esthetic, looks as if it is growing out of the gingiva
 - Difficult to achieve without ridge augmentation procedures or guided tissue healing following extractions

Intermediate abutment. When a tooth will serve as an abutment with edentulous spaces on either side, it requires special attention due to the class I lever mechanics of the fixed partial denture. A nonrigid connector is typically used on the distal of the intermediate abutment in this scenario.

Provisional restorations are critical in providing the patient with a temporary restoration during fabrication of the indirect restoration. They have many functions:

- Protects the pulp and periodontium
- Maintains arch integrity (prevents adjacent and/or opposing teeth from drifting)
- Provides function
- Confirms both esthetics and phonetics that is acceptable to the patient and dentist
- Can be used to check proper tooth reduction
- Cast of the provisionals provides the lab technician information about what the final restoration should look like

A comparison of the advantages and disadvantages of provisional restoration materials is provided in Table 3–6.

Dental Materials

Impression materials for fixed prosthodontics have changed over the years. Early materials included reversible hydrocolloid (agar) and polysulfide. Currently the market is dominated by addition silicones (polyvinylsiloxane [PVS]) and polyether.

Final Impression Materials

- **Reversible hydrocolloid** (Agar) is an impression material that changes from a gel to a sol with the application of heat and is reversible with cooling. Not used much anymore due to proliferation of elastomeric impression materials:
 - Ingredients:
 - Agar 12–15%
 - Potassium sulfate 1.7%
 - Water 85.5%

TABLE 3 – 6. Provisional Restoration Materials

MATERIAL	ADVANTAGES	DISADVANTAGES
PMMA	High strength Low cost Easily repaired	High exothermic setting reaction High shrinkage Bad odor
PEMA	Moderate strength Low cost Easily repaired Exothermic reaction higher than Bis-acryl, but lower than PMMA	Easily discolored Weaker than PMMA
Bis-acryl	Low exothermic setting reaction Minimal shrinkage Syringe delivery system Best indicated in single unit cases	High cost Weak (breaks under moderate stress) Difficult to repair

TABLE 3 – 7. Condensation Silicone

	BASE	CATALYST
Dimethyl siloxane	Alkyl silicate or ethyl ortho-silicate (cross linking agent)	
Fillers	Tin octoate (activator)	

- Disadvantages:
 - Needs special equipment
 - Must be poured immediately
 - Can only poured once
 - Poor tear strength
- **Polysulfide** (refer to section on complete denture impressions):
- **Condensation silicone** is an elastomeric impression material that sets in a cross-linking polymerization reaction and gives off the by-product ethanol (see Table 3–7).
- Disadvantages:
 - Must be poured immediately
 - Poor dimensional stability due to evaporation of ethanol
 - Hydrophobic
 - Low tear strength
- **Addition silicone** or PVS has become one of the most popular crown and bridge impression material (see Table 3–8.). There are no by-products compared to condensation silicones.
- Advantages:
 - Excellent dimensional stability (up to 2 weeks)
 - Excellent surface detail
 - Low permanent deformation
- Disadvantages:
 - Hydrophobic
 - Temperature sensitive
- **Polyether** is an elastomeric impression material (see Table 3–9).
- Advantages:
 - Excellent dimensional stability
 - Low permanent deformation
 - Hydrophilic
- Disadvantages:
 - Some can be very rigid and difficult to remove
 - Can absorb water



Latex will retard the setting of PVS.

TABLE 3 – 8. Addition Silicone/Polyvinylsiloxane

	BASE	CATALYST
	Silicone with terminal silane hydrogen groups	Silicone with terminal vinyl groups
Filler		Chloroplatinic acid
		Filler



Relief of 20–40 μm is desired for cement space.



The CTE of the porcelain must be compatible with that of the metal. Metal CTE should be slightly higher than the porcelain CTE.

TABLE 3–9. Polyether

BASE	CATALYST
Polyether (ethylene imine groups)	Sulfonate (aromatic sulfonic acid ester)
Silica filler	Thickening agent
Plasticizer	

Steps to fabricate wax pattern:

- Once the final impression is made, a master cast is fabricated in type IV die stone.
- The master cast can have a separate die(s) or a removable die(s).
- A **die** is a replica of the tooth preparation used to fabricate the restoration.
- The die(s) is/are trimmed (ditched) to allow the laboratory technician to:
 - fabricate the wax pattern
 - properly contour the axial surfaces with the proper emergence profile
 - finish the margins
- Die hardener—strengthens the margins to prevent abrasion of the die stone.
- Die spacer—applied to the die except the margins to provide relief for cement space.

There are many metals that are used for fixed prosthodontics. Most are alloys, which are mixtures of metals and nonmetals.

Metals

Properties of metals that are important:

- Strength (MPa)—compressive, tensile, and shear:
 - Ultimate tensile strength—maximum stress before fracturing
 - Yield strength—most important, resistance to permanent deformation
- Modulus (GPa)—rigidity or stiffness
- Hardness (kg/mm^2)—important for wear characteristics and finishing
- Porcelain–metal bond—chemical and microchemical bond formed by oxides on the surface of the alloy
- Coefficient of thermal expansion (CTE)—the change in length for a 1°C change in temperature—important for porcelain–metal bond
- Work hardening—the process of
- Phase structure—single phase vs. multiple phase
- Corrosion—release of elements; usually very low, but can occur

Allergies to metals are possible for some patients. Chronic inflammation around a crown margin with no other etiologic factor could be the result of metal allergy.

Noble metals: gold (Au), platinum (Pt), palladium (Pd), rhodium (Rh), ruthenium (Ru), iridium (Ir), osmium (Os)

- Corrosion resistant
- Oxidation resistant
- Higher cost than base metals

ADA classification of dental alloys:

1. High noble: $\geq 60\%$ noble and $\geq 40\%$ Au
 - Au-Pt
 - Au-Cu-Ag-Pd
 - Au-Pd-Ag
2. Noble: $\geq 25\%$ noble
 - Au-Cu-Ag-Pd
 - Pd-Cu-Ga
 - Ag-Pd or Pd-Ag
3. Base: $\geq 25\%$ noble (Ni, Cr, Be, Ti, Fe, Ag)
 - Ni-Cr
 - Co-Cr
 - Ti

Classification of dental alloys:

1. Type I—soft—inlays
2. Type II—medium—inlays/onlays
3. Type III—hard—crowns
4. Type IV—extra hard—RPDs

Investing and Casting Metals

- Wax pattern is made on the die
- Sprue is attached to wax pattern and then placed into a casting ring (sometimes used with a ring liner) and invested
- Investment material:
 - Gypsum-bonded investment:
 - 55–75% silica refractory, 25–45% gypsum binder, 5% modifiers
 - Used with low-heat metals
 - Three types of expansion → to compensate for shrinkage of metal:
 - Setting expansion when exposed to air
 - Hygroscopic expansion in water
 - Thermal expansion to heat
 - Phosphate bonded investment:
 - 80% quartz and/or cristobalite refractory, 20% MgO and monoammonium phosphate binder
 - Special liquid-containing colloidal silica:
 - Increases strength
 - Increases setting expansion
 - Decreases surface roughness
 - Used with high-heat metals
- Once investment is complete the wax is eliminated leaving a negative reproduction of the pattern (lost wax technique)
- Casting machine sends melted alloy into the investment space producing a replica of the wax pattern

Solder

- Soldering is the process of joining two pieces of metal together through the use of a lower fusing intermediate metal.
- Soldering can be done before (presolder) the porcelain is added or after the porcelain is finished (postsolder).



Nickel is the most common metal allergy, especially in females (10–20%).

- Metal frameworks for FPDs that do not fit well in the mouth can be sectioned and indexed in the mouth and then soldered.
- Fusion temperature of solder should be 100–150°F less than that of the alloy.
- Optimal space for solder is 0.25 mm, but better to be a bit bigger than too small.
- Flux (e.g., borax) is added to the metal surface to improve flow of the solder, dissolve oxides, and prevent contamination.
- Antiflux (e.g., graphite) is placed around the area that is to be soldered to restrict the flow.

Ceramics have come a long way in dentistry to the point now that an all ceramic restoration can mimic and be as esthetic as a natural tooth. This requires tremendous skill on the part of the dentist and laboratory technician/dental technologist, but it can be achieved.

Types of ceramics:

- Metal–ceramic systems:
 - Feldspathic (moderate leucite):
- All-ceramic systems:
 - Feldspathic (high leucite)
 - Powder
 - Pressed
 - Feldspathic (no leucite)—veneering ceramic
 - Glass ceramics (pressed and cast):
 - Micaeous
 - Lithium disilicate
 - Core ceramics:
 - Alumina
 - Spinel
 - Zirconia

Components of dental porcelain:

- Feldspar—acts as a matrix
- Quartz/silica (SiO_2)—provides skeleton and strength
- Kaolin—a clay that acts as a binder
- Fluxes or glass modifiers

Properties of dental porcelain:

- Low plastic deformation → brittle material
- Compressive strength is much greater than tensile or shear strengths

Metal–ceramic restorations:

- Before the porcelain is added to the metal framework, the metal must be taken through a degassing step, which is the process of removing any impurities and forming an oxide layer for the porcelain–metal bond.
- Opaque porcelain is first applied to the metal to mask the color.
- Then a layering process of different dentin and enamel porcelains is used in conjunction with different modifiers in several steps to create to final product.
- Shrinkage during firing = 20%.
- The last step is the glaze firing.
- Polishing the porcelain produces a surface as smooth as glazing.

All-ceramic methods of fabrication:

- Pressed ceramics (e.g., Empress)
- Cast ceramics (e.g., Dicor)
- Computer-aided design and computer-aided machining (CAD–CAM) (e.g., Procera, Lava, In-Ceram, and Cercon)

Ceramic cores:

- The all-ceramic market is moving toward the fabrication of cores using CAD–CAM techniques.
- Two main categories exist between alumina and zirconia
 - Main difference—zirconia is stronger, but alumina is more esthetic, because zirconia is very opaque

Color in dentistry is important for matching dental restorations to the patient's natural teeth.

Three dimensions of color:

1. Hue—the actual color (e.g., red, yellow, green, etc.)
2. Value—the relative lightness/whiteness or darkness/blackness; brightness
3. Chroma—the amount of saturation of the hue

Metamerism is when two objects appear to color match under one light source, but not another. Therefore, it is important to check the shade under two or more different light sources.



The most important dimension of color for dental restorations is value.

INSERTION AND POSTINSERTION

Cementation—the purpose of the cement or luting agent is to fill in the space between the tooth and the indirect restoration.

There is no one ideal luting agent in dentistry. Each cement has its indications and contraindications. Some ideal properties for cements include:

- Biologically compatible
- Good handling properties including sufficient working time and quick setting time
- Low viscosity and film thickness
- Low solubility to saliva, water, and acid
- High strength
- Adhesion to both tooth structure and restoration
- Bacteriocidal and/or cariostatic properties
- Good esthetics including translucency

Types of cements:

1. **Zinc oxide eugenol:**
 - a. Considered a permanent cement, but poor strength properties, so no longer used for final cementation.
 - b. Ethyl benzoid acid (EBA) can be added to increase strength
 - c. IRM is a ZOE with PMMA beads added to the powder to increase strength

Powder	Liquid
ZnO 69%	Eugenol 85%
Rosin	Olive oil 15%
Zn stearate and acetate	
EBA adds the following:	
Al ₂ O ₃	<i>o</i> -Ethoxybenzoic acid
Advantages	Disadvantages
Biocompatible and low pulpal irritation (sedative effect)	Inhibits polymerization of PMMA Low strength
Good working time	High solubility
Bactericidal	

2. Zinc phosphate:

- Working time can be varied by use of a cool glass slab with incremental mixing
- Increase in powder → increase in strength
- Exothermic reaction → mixing on wide area of glass slab to dissipate heat
- Film thickness—25 μm

Powder	Liquid
ZnO 90%	H ₃ PO ₄ 38%
MgO assists calcinations process	H ₂ O 32%
SiO ₂ and Bi ₂ O ₃ increases smoothness	AL + Zn added to slow down reaction
Advantages	Disadvantages
Easy to use	Pulp irritation due to initial acidity
Low cost	Solubility to oral fluids
Easy clean-up	No chemical bond
Excellent track record historically	

3. **Zinc polycarboxylate:**
 - a. Mixing ratio: 3 drops of liquid to 1 scoop of powder
 - b. Use only if mix is still glossy

Powder	Liquid
ZnO	Polyacrylic acid 40%
MgO	
Al ₂ O ₃	
Advantages	Disadvantages
Neutral pH	Short working time
Adhesion to tooth structure	Strength not as high

4. **Glass ionomer:**
 - a. Setting reaction has two stages
 - Ca²⁺ binds two –COOH to form a soluble gel
 - Al³⁺ replaces Ca²⁺ and binds three –COOH to form the insoluble cement

Powder	Liquid
SiO ₂ (silicate glass)	Polyacrylic or polymaleic/itaconic acid
Al ₂ O ₃ + CaF ₂	
Na ₃ AlF ₆ + AlF ₃ + AlPO ₄	Tartaric acid
Fluoride	
Advantages	Disadvantages
High strength	Sensitive to moisture in early stages
Adhesion to tooth structure	Difficult clean-up
Low solubility	Correct powder/liquid ratio important
Fluoride release	

5. **Resin-modified glass ionomer:**
 - a. The addition of resin results in the following properties when compared to glass ionomer:
 - Decreased bond to dentin
 - Increased microleakage
 - Less sensitive to moisture
 - Increased strength
 - b. Most resin-modified glass ionomer cements now come in automix carpules, which eliminate mixing problems and variations, and is becoming the cement of choice for many situations.
6. **Resin:**
 - a. Types of resin cements:
 - Chemical-cure, light-cure, or dual-cure
 - Total-etch, self-etch, or self-adhesive
 - b. Some all-ceramic restorations can be bonded to tooth structure with the use of resin cements

Advantages	Disadvantages
High strength	Increased shrinkage
Low solubility	Technique sensitive
Adhesion	Difficult clean-up

Proper oral hygiene instructions are essential for long-term success.

- Cleaning under FPDs with superfloss or a floss threader

CHAPTER 4

Oral and Maxillofacial Surgery

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Jason E. Portnof, DMD, MD

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Local Anesthesia**ANATOMY AND TECHNIQUE**

Trigeminal nerve (cranial nerve V) (see Table 4–1)

- Originates in Pons.
- Both motor and sensory nerve.
- Three large trunks originate from the semilunar ganglion (Gasserian ganglion) (see Figs. 4–1, 4–2, 4–3, and 4–4).

REGIONAL ANESTHESIA IN DENTISTRY

- Anesthesia of the maxilla
 - Local infiltration
 - Buccal vestibule for posterior, middle, and anterior superior alveolar nerves
 - Infraorbital nerve block
 - Anesthetizes maxillary anterior teeth
 - Supplementary injection of the palate
 - Nasopalatine nerve block
 - Greater palatine nerve block
- Anesthesia of the mandible
 - Mandibular nerve block
 - Direct method
 - Gow gates mandibular nerve block
 - Akinosi (closed mouth) mandibular nerve block
 - Mental nerve block
 - Lingual nerve block
 - Buccal nerve block

TABLE 4–1. Trigeminal Nerve Branches

TRIGEMINAL NERVE (V)		
OPHTHALMIC NERVE (V1)	MAXILLARY NERVE (V2)	MANDIBULAR NERVE (V3)
Nasociliary nerve	Zygomatic nerve	Auriculotemporal nerve
Supraorbital nerve	Posterior superior alveolar nerve	Lingual nerve
Lacrimal nerve	Middle superior alveolar nerve	Buccal nerve
Frontal nerve	Anterior superior alveolar nerve	Inferior alveolar nerve
Supratrochlear nerve	Infraorbital nerve	(mental nerve)
Infratrochlear nerve	Greater palatine nerve	
	Nasopalatine nerve	

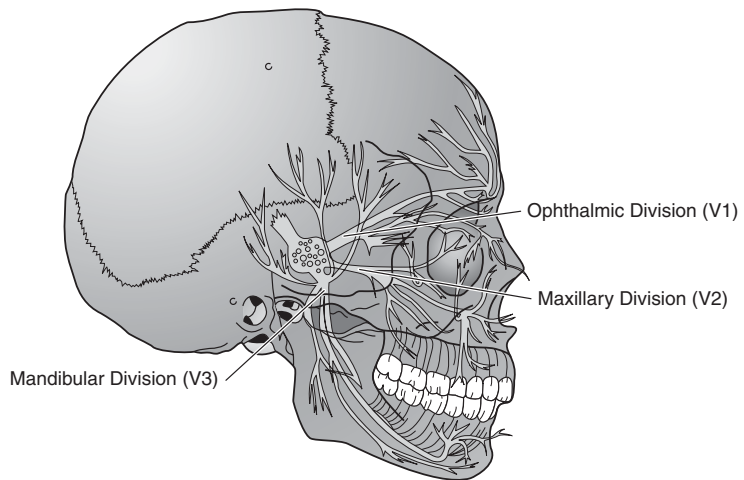


FIGURE 4-1. Trigeminal nerve distribution.

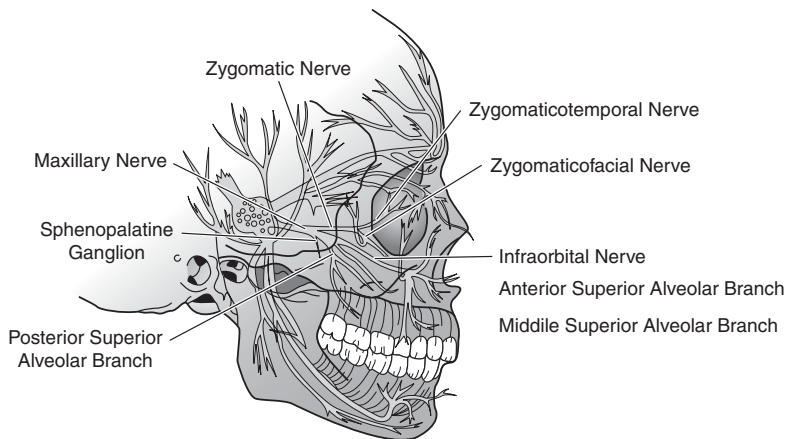


FIGURE 4-2. Maxillary nerve.

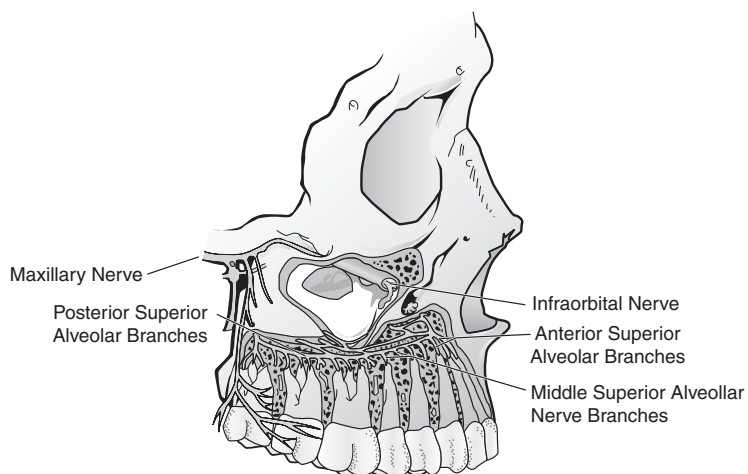


FIGURE 4-3. Maxillary nerve and its distribution.

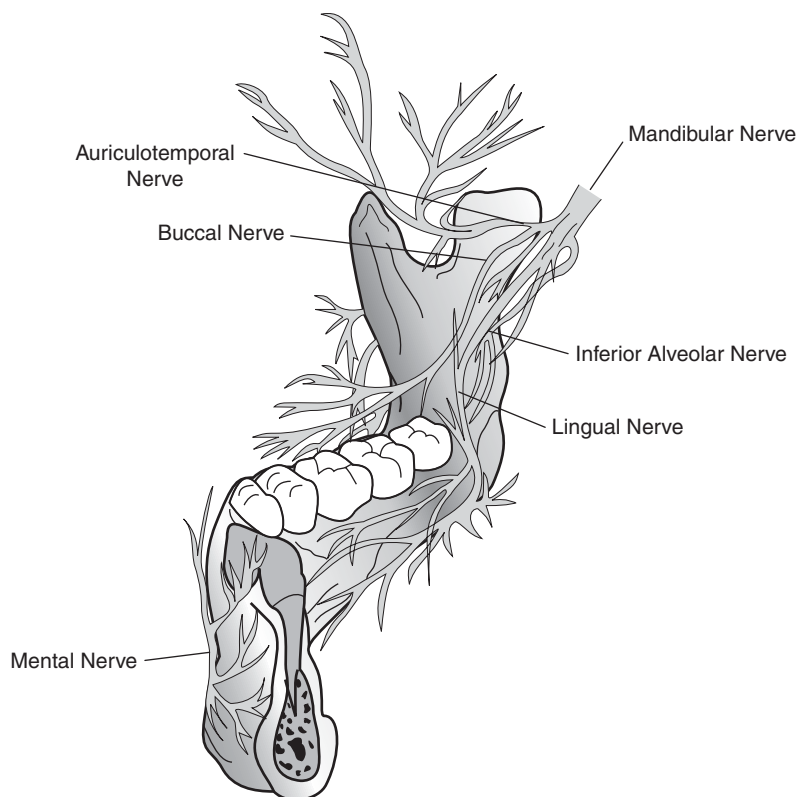


FIGURE 4-4. The mandibular nerve and its distributions.

Clinical Pharmacology

CLASSIFICATION OF LOCAL ANESTHETICS (LAs)

- All LAs are comprised of a lipophilic aromatic ring linked to a hydrophilic amino group.
- This link is either an ester or an amide bond and determines classification (see Fig. 4-5).

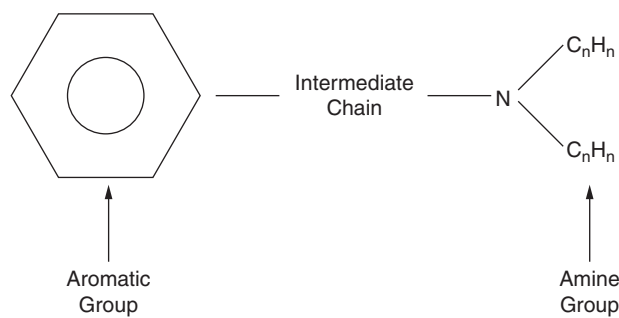


FIGURE 4-5. Local anesthetic structure.

Amides	Esters
■ Lidocaine ■ Mepivacaine ■ Bupivacaine	■ Novocaine ■ Procaine ■ Benzocaine ■ Tetracaine
Metabolized by the microsomal P-450 enzymes of the liver. ■ N-dealkylation ■ Hydroxylation	Metabolized by pseudocholinesterase in the plasma.

DOSAGES (ADULT) FOR LOCAL ANESTHETICS

Dosages are based on average weight of 150 lb or 70 kg (see Table 4–2)

CLARK’S RULE FOR PEDIATRIC DOSING OF LOCAL ANESTHETICS

- Maximum pediatric dose = (weight of child in lb./150) (maximum adult dose in mg)

TABLE 4–2. Dosages (Adult) for Local Anesthetics (based on average weight of 150 lbs or 70 kg)

AGENT	MG/CARTRIDGE	MAXIMUM DOSE	MAXIMUM DOSE
		MG/KG	MG
2% lidocaine (Xylocaine)	36	4.5	300
2% lidocaine with 1:100,000 epinephrine	36	7	500
3% mepivacaine (Carbocaine, Polocaine)	54	5.5	400
2% mepivacaine with 1:20,000 levonordefrin	36	5.5	400
4% prilocaine (Citanest)	72	8	600
4% prilocaine with 1:200,000 epinephrine	72	8	600
0.5% bupivacaine with 1:200,000 epinephrine (Marcaine)	9	1.3	90
1.5% etidocaine with 1:200,000 epinephrine (Duranest)	27	5.5	400
4% articaine with 1:100,000 epinephrine (Septocaine)	68	7	500



*All local anesthetics generally have the letter **I** plus **caine** in their drug names. (Lidocaine, mepivacaine, bupivacaine)*



*Lidocaine is metabolized by the **Liver**. (Both words contain the letter **L**)*



The maximum amount of 2% lidocaine with 1:100,000 epinephrine that can be administered to a healthy 150 lb man is 477 mg (~ 13 dental cartridges).



Conversion of lb to kg: (2.2 lb/kg).



Adding a vasoconstrictor (like epinephrine) to the local anesthetic decreases its rate of absorption. This increases its duration of action, minimizes systemic toxicity, and helps with hemostasis.

Each 1.8 cc cartridge of 2% lidocaine with 1:100,000 epinephrine contains:

- 20 mg/cc of lidocaine
- 36 mg of lidocaine
- 0.018 mg of epinephrine



It is generally safe to use 1 cartridge for every 20 pounds of 2% lidocaine with 1:100,000 epinephrine in the healthy pediatric patient.



Infection causes tissue to become acidic. This results in an increase in the ionized form of the local anesthetic at the expense of the unionized form of the local anesthetic.

$$RNH_3 + H^+ \rightleftharpoons RH_4^+$$

This prevents passage of the LA through the nerve membrane, decreasing the effectiveness of the LA!

MECHANISM OF ACTION OF LOCAL ANESTHESIA

Physiology of the peripheral nerve

- An inactive nerve cell has a resting membrane potential of -50 to -70 mV.
- The cytoplasm of a resting nerve cell has a high concentration of potassium ions (K^+) and a low concentration of sodium ions (Na^+). Maintained by the Na/K ATPase pump.
- At rest, the cell membrane is relatively resistant to ion passage.
- Excitation causes a transmembrane action potential, and the cell membrane's ion permeability increases.

Phases of nerve action potential (AP) (see Fig. 4–6)

- Excitation:
 - Requires stimulus between -90 mV and 60 mV.
- Slow phase of depolarization:
 - Influx of sodium ions through Na^+ channels.
 - Electrical potential within the nerve cell becomes less negative.
- Threshold potential is reached:
 - Potential difference between the outside and the inside of the nerve cell reaches a critical level.
 - Depolarization reverses the cell's potential so that nerve interior is positively charged when compared to the exterior of the cell membrane.
 - At the peak of the AP, the intracellular positive potential reaches approximately $+40$ mV.
- Repolarization:
 - Potassium ions pass out of the cell.
 - Restoration of intracellular resting potential of -50 to -70 mV.

Blocking of nerve conduction

- When injected into the tissue, the LA exists in both ionized and nonionized forms.
- Then nonionized base penetrates tissue readily.
 - The LA's lipophilic aromatic ring facilitates passage through the nerve sheath and membrane.

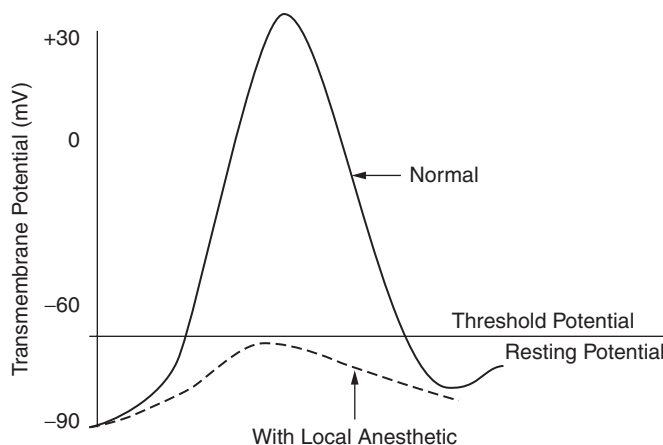


FIGURE 4–6. Nerve action potential graph.

- Reequilibration between nonionized and ionized forms occurs within the nerve cell.
- The ionized form inhibits the nerve cell membrane's sodium channels.
 - Preventing inflow of Na^+ .
 - Rate of depolarization is slowed.
 - Threshold potential is not reached.
 - Preventing formation of action potentials.

(See Fig. 4–7A and B)

COMPLICATIONS

- LA toxicity:
 - Due to elevated plasma levels of LA.
 - Inadvertent intravascular injection of LA.
 - Violation of maximum mg/kg dose of LA.
 - Signs of LA toxicity:
 - Circumoral numbness
 - Initial signs (cardiovascular and central nervous systems):
 - Tachycardia
 - Hypertension
 - Drowsiness
 - Confusion
 - Tinnitus
 - Metallic taste
 - Later signs:
 - Tremor
 - Hallucinations
 - Hypotension
 - Bradycardia
 - Decreased cardiac output
 - Latest signs:
 - Unconsciousness
 - Seizures
 - Ventricular dysrhythmias
 - Respiratory and circulatory arrest
- Allergy:
 - True hypersensitivity reactions to LA are rare.
 - Esters
 - Derivatives of *p*-aminobenzoic acid (PABA).
 - More likely to induce allergic reactions than amides.
 - Methylparaben:
 - Bacteriostatic preservative.
 - May be the causative agent in many hypersensitivity reactions.
- Methemoglobinemia:
 - Hemoglobin is oxidized to methemoglobin.
 - Methemoglobin cannot bind and carry oxygen.
 - Caused by excessive doses of benzocaine, prilocaine, or lidocaine.
 - Clinical signs: decreased pulse oximetry, cyanosis, “chocolate-colored” blood visible in the surgical field.
 - Treatment: IV methylene blue (1–2 mg/kg of 1% solution over 5 minutes).



LA can be mixed with sodium bicarbonate to alkalize the solution in order to decrease pain upon infiltration and increase effectiveness.



Small, unmyelinated nerve fibers (pain, temperature, autonomics) are more sensitive to local anesthetics than larger, myelinated nerve fibers.



Following anesthetic injection, sensation will be lost in the following order: pain → cold → warm → touch → deep pressure → motor.



Seizures are the most common adverse effect resulting from systemic absorption of toxic amounts of local anesthetics.

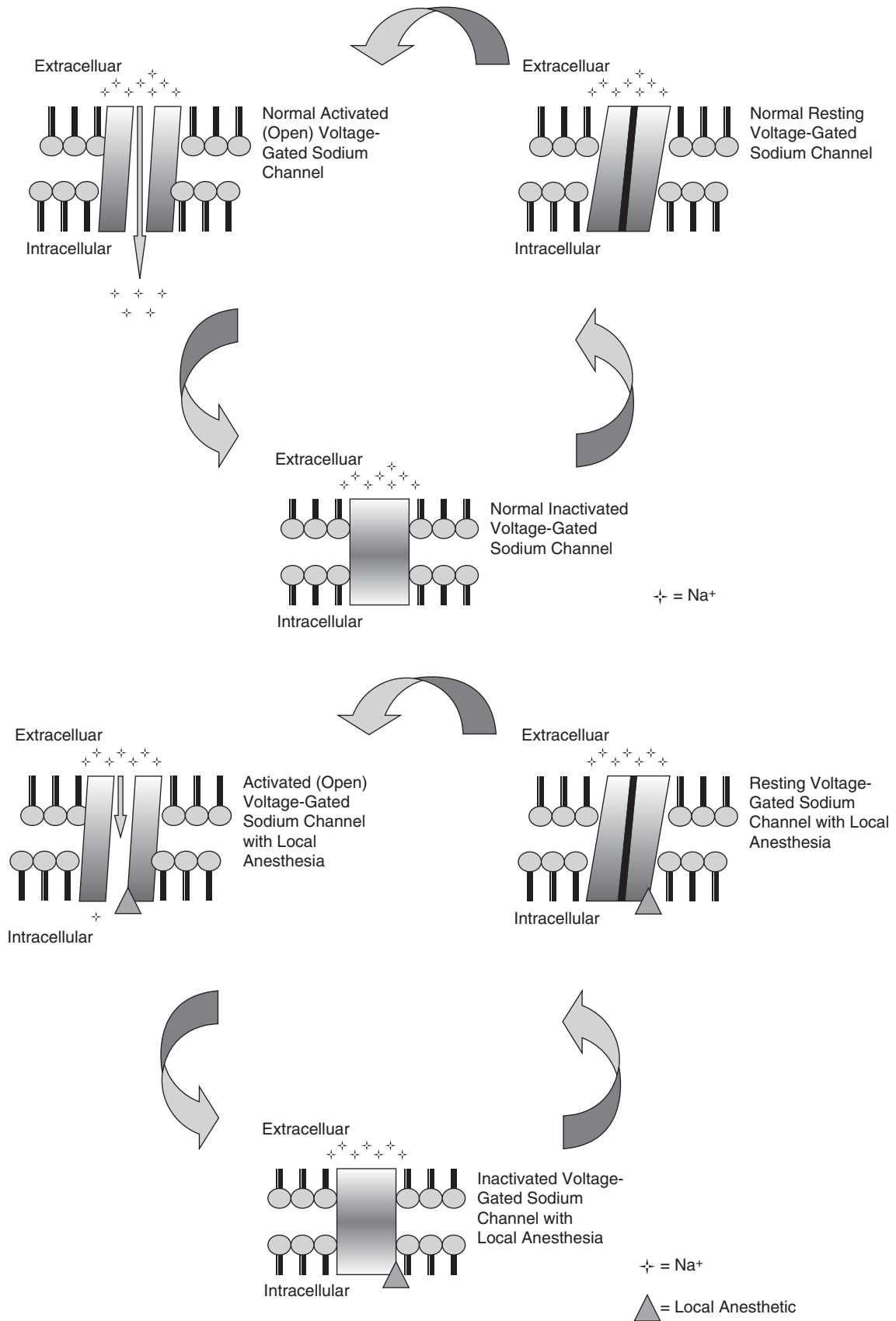


FIGURE 4-7. Voltage-gated sodium channels.

TABLE 4-3. American Society of Anesthesiologists (ASA) Physical Status Classification

P1	A normal healthy patient
P2	A patient with mild systemic disease
P3	A patient with severe systemic disease
P4	A patient with severe systemic disease that is a constant threat to life
P5	A moribund patient who is not expected to survive without the operation
P6	A declared brain-dead patient whose organs are being removed for donor purposes

Note: If the procedure is an emergency, the physical status is followed by "E" (i.e., 3E).

CONSCIOUS SEDATION

Choosing the appropriate sedation candidate: Most office-based anesthesia is performed on patients with preoperative classifications of P1 or P2 (see Table 4-3).

Guedel's Signs and Stages of Anesthesia

Stages	Description
Amnesia and analgesia	Inducement, preservation of protective reflexes, stage ends with loss of consciousness
Delirium and excitement	Involuntary movements, obtunded reflexes, stage ends with onset of total anesthesia (Nausea and vomiting are common in this stage.)
Surgical anesthesia	Three planes Light Medium (ideal plane for invasive surgical procedures) Deep
Medullary paralysis	Very deep anesthesia with loss of cardiovascular function and imminent death

INHALATION ANESTHETICS (SEE TABLE 4-4)

Anesthesia uptake is based on:

- Solubility
 - Drugs with decreased blood solubility have rapid induction and recovery times.
 - Drugs with increased lipid solubility have increased potency (1/MAC).
- Alveolar blood flow
- Difference in partial pressure between alveolar gas and venous blood (gradient).



Children and the elderly are at greatest risk for local anesthetic toxicity.



Anesthesia consists of a continuum of stages with overlapping boundaries.



Conscious sedation falls under stage 1 of the anesthesia stages. For the safety of your patient stay within this stage!



Vomiting during stage 2 of anesthesia may lead to aspiration. Remember ABCs!



Nitrous is the least potent of the inhalation agents.

TABLE 4-4. Properties of Inhalation Anesthetics

INHALATION ANESTHETIC	PHASE AT ROOM TEMPERATURE	MAC (%)	BLOOD-GAS PARTITION COEFFICIENT
Nitrous oxide	Gas	104	0.47
Isoflurane	Volatile liquid	1.2	1.4
Halothane	Volatile liquid	0.75	2.3
Desflurane	Volatile liquid	6.0	0.42
Sevoflurane	Volatile liquid	1.7	0.68
Enflurane	Volatile liquid	1.6	1.9

Minimum alveolar concentration (MAC):

- The amount of drug necessary to prevent movement in 50% of patients subjected to a standardized stimulus (i.e., surgical incision) at 1 atmosphere.

Blood-gas partition coefficient:

- The difference between the partial pressures of a gas and blood indicates how quickly the agent crosses the pulmonary membrane and enters the bloodstream.
- The higher the value, the higher the solubility.

Nitrous oxide:

- “Laughing gas.”
- Minimum 30% O₂ delivery.
- Potent analgesic.
- Weak general anesthetic.
- No biotransformation.
- Side effects: Headaches, nausea/vomiting, lethargy, and diffusion hypoxia.

Diffusion hypoxia:

- May occur if the patient is permitted to recover from nitrous oxide sedation while breathing in room air.
- Nitrous oxide from the bloodstream diffuses into the alveoli in the lungs for elimination. This mixes with inhaled room air which contains 20% O₂, resulting in hypoxia.
- Is prevented by administering high-concentration oxygen during the recover period of nitrous sedation.

Occupational risk:

- Prolonged exposure to nitrous oxide
 - Bone marrow suppression
 - Megaloblastic anemia
 - Leukopenia

- Neurological deficiencies
 - Peripheral neuropathies
 - Pernicious anemia

INTRAVENOUS/ENTERAL ANESTHETICS

- Barbituates:
 - Act as sedatives and hypnotics.
 - Potent anesthetics.
 - Weak analgesics.
 - Depress central nervous system (CNS) neuronal activity by decreasing rate of dissociation of gamma-aminobutyric acid (GABA) at its receptor.
 - Increases *duration* of chloride channel opening, thus decreasing neuronal firing.
 - Prolonging GABA's inhibitory effect in the reticular activating system (RAS).
 - Thiopental sodium (Pentothal)
 - Ultra-short-acting agent
 - High lipid solubility
 - Crosses blood–brain barrier quickly.
 - Methohexital (Brevital)
 - Ultra-short-acting agent
 - Phenobarbital (luminal)
 - Long-acting agent
- Benzodiazepines:
 - Act as:
 - Anxiolytic
 - Anticonvulsant
 - Antispasmodic
 - Sedative/hypnotic
 - Amnesic (anterograde amnesia)
 - Enhance the binding of GABA to the GABA receptors complex.
 - Increases *frequency* of chloride channel opening, thus decreasing neuronal firing.
 - Available in PO (by mouth) and IV (intravenous) forms.
 - Duration of effect



The risk of respiratory depression and coma is less for benzodiazepines than for barbiturates.

Short-Acting <6 hours	Intermediate-Acting Between 6 and 10 hours	Long-Acting >10 hours
■ Triazolam (Halcion) PO ■ Midazolam (Versed) PO, IV	■ Alprazolam (Xanax) PO	■ Diazepam (Valium) PO, IV ■ Lorazepam (Ativan) PO, IV ■ Chlordiazepoxide (Librium) PO, IV



The effect of flumazenil lasts only for about 20 minutes. . . .

Beware of resedation!



Propofol is good for outpatient anesthesia due to its rapid induction and recovery, and lower incidence of nausea and vomiting.



Propofol is nicknamed the "Milk of Amnesia."



Ketamine produces a sedation in which the patient appears awake, but is unconscious, and does not feel pain.



Opioid withdrawal is not life threatening, unlike alcohol or benzodiazepine withdrawals.

- Flumazenil:
 - Used to reverse the effects of benzodiazepines.
 - Competitive antagonist at GABA receptor.
- Propofol (Diprivan):
 - IV Sedative-hypnotic agent
 - Highly lipophilic
- Ketamine:
 - Phencyclidine (PCP) derivative.
 - NMDA receptor antagonist.
 - Short acting.
 - Produces dissociative anesthesia.
 - Dissociation between thalamic and limbic systems.
 - Cardiovascular stimulant.
 - Often used in children and young adults.
 - Can cause postoperative disorientation/hallucinations.
- Chloral hydrate:
 - Frequently used in children.
 - Sedative and hypnotic: CNS depressant.
 - Active metabolite: Trichloroethanol.
 - Onset of action: 30 minutes to 1 hour.
 - Duration: 4–8 hours.
 - Contraindicated in hepatic and renal impaired patients.
 - Toxicity:
 - Hypotension
 - Respiratory depression
 - Hypothermia
 - Cardiac arrhythmias
 - Coma
- Opioids:
 - Narcotics
 - Act as agonist on mu (μ), delta (δ), kappa (κ), and sigma (σ) receptors in the CNS
 - μ receptors (supraspinal)
 - Responsible for analgesia and euphoria
 - Available in both PO and IV forms:
 - Fentanyl (Sublimaze) IV, PO (lozenges)
 - Sufentanil (Sufenta) IV
 - Alfentanil (Alfenta) IV
 - Morphine IV, PO
 - Codeine PO
 - Meperidine (Demerol) IV, PO
- Opioids adverse effects:
 - Pruritis (due to histamine release)
 - Nausea/vomiting
 - Urinary retention
 - Constipation
 - Miosis
 - Respiratory depression
- Signs of opioid withdrawal:
 - Hypertension
 - Piloerection, chills ("quitting cold turkey")
 - Sweating

- Nausea/vomiting
- Abdominal cramping
- Restlessness (“kicking the habit”)
- Mydriasis
- Lacrimation and rhinorrhea
- Insomnia
- Naloxone:
 - Pure mu-receptor antagonist.
 - Used to reverse the effects of opioids.



Naloxone also reverses opioids analgesic effect. Be careful when using in chronic pain patients! Can precipitate opioid withdrawal!

COMPLICATIONS

- Malignant hyperthermia:
 - Rare inherited disease.
 - Prevents the release of calcium from the sarcoplasmic reticulum of skeletal muscle leading to persistent contraction.
 - Triggering agents: Succinylcholine and inhalation anesthetics (Halothane).
 - Signs/symptoms: Rigidity, fever, tachycardia, metabolic acidosis, hypercarbia, and hypoxia.
 - Treatment: Dantrolene.
- Phlebitis:
 - Inflammation of a superficial vein.
 - Can occur after insertion of a venous catheter for administration of drugs.
 - Signs/symptoms: Pain, tenderness, induration, and/or erythema.
 - Treatment: Elevation of limb, moist heat, nonsteroidal anti-inflammatory drugs (NSAIDs).
- Laryngospasm:
 - Forceful involuntary spasm of laryngeal musculature.
 - Can be caused by oral fluids triggering the laryngeal reflex during lighter stages of anesthesia.
 - Can be prevented by using a pharyngeal barrier and tonsil suction.
 - Treatment: Positive pressure oxygen-supplemented ventilation via face-mask.
 - If persistent: Succinylcholine.
 - Last resort: Cricothyroidotomy.



The most common medical emergency encountered in the dental office is syncope.



Most common complication of office-based anesthesia is a loss of airway. Remember ABCs!

Medical assessment and emergency care.
Vital signs:

1. Temperature (Normal oral temperature is 98.6°F or 37°C.)
2. Heart rate (Normal range is 60–80 beats per minute.)
3. Blood pressure (Normal is approximately 120/80 mm Hg.)
4. Respiratory rate (Normal range is 12–18 breaths per minute.)

Basic Life Support

CARDIOPULMONARY RESUSCITATION

ABCDs of CPR:

- Airway:
 - Head tilt
 - Jaw thrust (if neck trauma suspected)



The first step when initiating CPR is to establish unresponsiveness.



The time interval from cardiac arrest to first defibrillation is the most important factor in determining survival.

For every minute delay in defibrillation from the time of collapse, patient survival declines by 7–10% per minute.

- Breathing:
 - Look, listen, feel
 - If respirations are absent/inadequate, then provide rescue breathing.
 - Bag valve mask (BVM)
 - Ventilation rate: 1 breath every 5–6 seconds (10–12 breaths per minute)
- Circulation:
 - Check pulse
 - If pulse is absent, then initiate chest compressions.
 - During CPR, the compression-to-ventilation ratio is 30:2 (rate of 100 compressions per minute).
- Defibrillation:
 - Automated external defibrillator

► ORAL SURGERY

Wound Healing (Table 4–5)

Methods of wound healing:

- Primary intention:
 - Closely reapproximated wound edges.
 - Lower risk of infection.
 - Minimal scar formation.
 - Examples: well-approximated surgical incision, well-reduced fracture.
- Secondary intention:
 - Large gap between the incision edges.
 - Requires larger amount of epithelial migration, collagen deposition, contraction, and remodeling (granulation tissue).
 - Healing is slower.
 - More scar formation.
 - Examples: extraction socket, ulcers, dehiscent wound.

Factors that impair wound healing:

- Foreign material
- Necrotic tissue
- Ischemia
- Tension
- Systemic conditions (e.g., diabetes, leukemia, steroid therapy)



Scar tissue is weaker than the original tissue.

TABLE 4–5. Stages of Wound Healing

STAGE OF WOUND HEALING	INFLAMMATORY STAGE	PROLIFERATIVE STAGE	REMODELING/MATURATION STAGE
Time period	Immediate to 2–5 days	2 days to 3 weeks	3 weeks to 2 years
Description	Hemostasis ■ Vasoconstriction ■ Clot formation Inflammation ■ Vasodilation ■ Phagocytosis	Epithelialization Angiogenesis Granulation tissue formation Collagen deposition	Collagen fibers to increase tensile strength Contraction occurs

Dentoalveolar

Indications for tooth extraction:

- Severe caries (nonrestorable tooth).
- Pulpal necrosis:
 - RCT failure.
 - Tooth not a candidate for endodontic treatment.
- Severe periodontal disease.
- Combined endodontic-periodontic lesion.
- Tooth malposition/eruption disturbances:
 - Impaction.
 - Crowded arch.
 - Overeruption.
- Supernumerary teeth.
- Teeth associated with pathology.
- Fractured tooth (nonrestorable).
- Preprosthetic extractions.
- Orthodontic indications.
- Medical indications:
 - Preradiation therapy (XRT).
 - Precardiac surgery (particularly valve replacement or valve repair).
 - Pretransplantation surgery.
 - Prebisphosphonate therapy (especially IV bisphosphonates).

Contraindications for elective tooth extraction:

- Systemic:
 - Uncontrolled diabetes.
 - End-stage renal disease with severe uremia.
 - Uncontrolled leukemia or lymphoma.
 - Uncontrolled cardiac disease:
 - Uncontrolled hypertension.
 - Unstable angina.
 - Recent MI.
 - Severe bleeding disorders:
 - Hemophilia.
 - Thrombocytopenia.
 - Pregnancy first and third trimester.
 - Patients taking certain medications.
 - IV bisphosphonates.
 - Chemotherapeutics agents.
 - Anticoagulants drugs.
 - Steroids.
- Local:
 - History of XRT.
 - Teeth within a tumor.
 - Severe pericoronitis.

Indications for extraction of impacted teeth:

- Prevention of:
 - Periodontal disease (periodontal pocket of 4 mm or more between the second molar and impacted third molar)
 - Pericoronitis (caused by normal oral flora, minor trauma, food impaction)
 - may lead to serious fascial space infections
 - Caries
 - Root resorption



The mesiodens is the most common supernumerary tooth.



Teeth within the line of jaw fracture are often salvaged if they do not interfere with reduction of the fractured bone segments, and are not severely damaged. In some instances, the tooth in the line of fracture helps to prevent displacement of the proximal segment of the mandibular fracture and these teeth should be retained to if a closed reduction is indicated. Teeth in the line of fracture may require removal in the future if infection occurs.



Impacted teeth should be assessed with the SLOB rule (same lingula, opposite buccal) or BAMA rule (buccal always moves away).



Patients should wait 6 months after CABG surgery prior to elective tooth extraction.



Extractions performed in areas that received XRT may lead to osteoradionecrosis.



Acute infection is not a contraindication to extraction.

Remember LA and pH principles.



The palatal root of the maxillary first molar is the most common root to be accidentally displaced into the maxillary sinus.

- Odontogenic cysts and tumors
- Fracture of the jaw
- Preorthodontic treatment

Complications of dentoalveolar surgery:

- Bleeding
- Infection
- Dry socket
- Aspiration of foreign objects or tooth
- Oral mucosa laceration
- Air emphysema (often caused by nonsurgical handpieces)
- Temporomandibular discomfort/injuries/trismus
- Jaw fracture (mandible, tuberosity, alveolar bone)
- Root fracture
- Damaged to adjacent teeth
- Oroantral communication
- Displacement of root fragments or teeth into:
 - Submandibular space
 - Mandibular canal
 - Maxillary sinus
 - Infratemporal fossa

Sequella of oroantral communication:

- Chronic oroantral fistula
- Maxillary sinusitis

Treatment of oroantral communication:

- <2 mm diameter → no surgical treatment.
- 2–6 mm → figure 8 suture to ensure maintain integrity of blood clot within the socket.
- >7 mm → requires flap closure.
- Sinus precautions (avoid nose blowing, sneezing, using straws, smoking).
- Nasal decongestants and antibiotics are recommended for oroantral communications >2 mm.

Hemostasis

Stages:

- Vasoconstriction of blood vessels
- Primary hemostasis
 - Platelet and collagen interaction leading to hemostatic plug
- Secondary hemostasis
 - Cascade of coagulation factors
- Repair process
 - Growth of fibroblasts and smooth muscle
 - Fibrinolysis/dissolution of the clot

How to achieve hemostasis:

- Pressure
- Suture ligation
- Electrocautery (thermal coagulation)
- Thrombin
- Tranxenamic acid
- Cellulose sheet (Surgicel)
- Gelatine sponge (Gelfoam)

- Bone wax (Beeswax and paraffin)
- Microfibrillar collagen (Avetene)

Oral Surgery Instruments/Armamentarium

- Aspirating syringe
- Blade:
 - #10
 - #11
 - #12
 - #15 (most commonly used for intraoral surgery)
- Periosteal elevator
- Elevators:
 - Straight
 - Curved
- Forceps
 - Maxillary universal forceps #150 → all maxillary teeth
 - Mandibular universal forceps #151 → all mandibular teeth
 - Cowhorn forceps → posterior mandibular molar teeth
 - Ash forceps → mandibular premolar
- Rongeurs
- Burr and handpiece
- Curettes

Sutures

- Suture sizes:
 - Diameter (# of Os)
 - Higher # of Os represents a smaller diameter suture
- Suture material
 - Absorbable:
 - Gut:
 - Plain
 - Chromic
 - Vicryl
 - Polydioxanone (PDS)
 - Nonabsorbable:
 - Silk
 - Nylon
 - Polypropylene (Prolene)
 - Mersilene (Dacron)
- Needles
 - Atraumatic:
 - Tapered point
 - Cutting
 - Reverse cutting
- Suture techniques:
 - Simple interrupted
 - Simple continuous
 - Mattress (horizontal or vertical)
 - Figure “8”
 - Continuous locking
 - Subcuticular



*Steps in extraction:
Recommended sequence for
extraction should be in the
following order:
maxillary → mandibular
posterior → anterior.*



*The first force to be applied
with a forceps should be
apical.*



*Bucco-lingual forces are less
effective in extracting
mandibular posterior teeth
due to the dense posterior
mandibular bone.*



*Normal saline (isotonic
solution) should be used for
surgical irrigation. Distilled
water is hypotonic, and will
cause cell lysis due to
differences in the osmotic
gradient.*

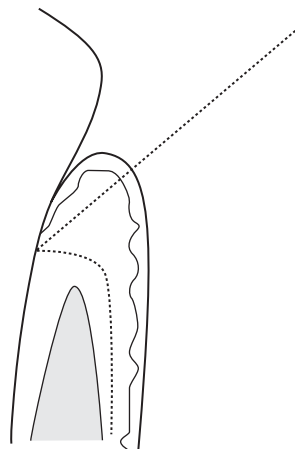


FIGURE 4-8A. Split-thickness mucoperiosteal flap.

FLAP DESIGN

- Incision should be made over sound bone.
 - Vertical release incision should be made at the line angle of the tooth, never on the facial aspect or splitting the papilla.
 - Should be designed with broad base to provide adequate blood supply to the free margin.
 - Should be adequate for visualization of the surgical field.
- Mucoperiosteal flap (see Fig. 4-8A and 4-8B):
 - Partial (split) thickness
 - Full thickness:
 - Envelope flap
 - Envelope flap with release

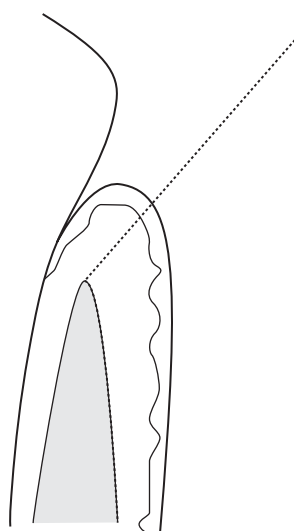


FIGURE 4-8B. Full-thickness mucoperiosteal flap.

NERVE INJURY

- Anesthesia: Loss of sensation.
- Paresthesia: Abnormal sensation (burning, tingling, pricking, or tickling).
- Hyperesthesia: Increase in sensitivity.
- Dysesthesia: Painful sensation to normal stimulus.
- Neurapraxia:
 - Mild injury with no axonal damage.
 - Spontaneous recovery within 4 weeks.
- Axonotmesis
 - Axonal damage but intact endoneural and perineural sheath (see Fig. 4–9).
 - Wallerian degeneration distal to injury.
 - Potential for recovery in 1–3 months.
- Neurotmesis:
 - Complete severance of axon with gap.
 - No recovery expected without surgery.

DRY SOCKET (ALVEOLAR OSTEITIS)

- Etiology:
 - Increased fibrinolytic activity.
 - Loss of blood clot.
 - Smoking and oral contraceptives have been implicated.
- Signs:
 - Worsening, throbbing pain.
 - Radiation.
 - Fetid odor.
 - Bad taste.
 - Poorly healed extraction site.

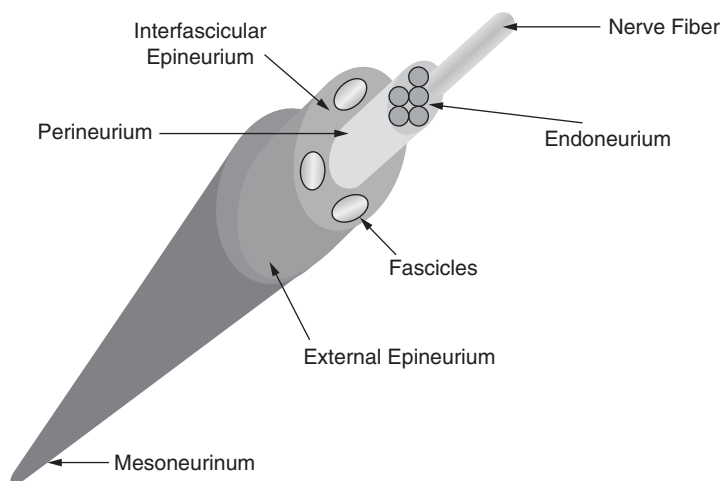


FIGURE 4–9. Normal anatomy of peripheral nerve subunits.



It is contraindicated to place implants immediately after the extraction of teeth in a region of active infection. Wait 8 weeks to place implants after extraction of infected teeth.



A cooling saline spray is used during bone preparation and implant placement to keep the temperature of the bone below 47°C.

- Treatment:
 - Irrigation with normal saline.
 - Sedative dressing (Eugenol-based).
 - To be changed every 48 hours until patient is asymptomatic.
 - Control pain (analgesic drugs).

► RECONSTRUCTIVE ORAL AND MAXILLOFACIAL SURGERY

Implant Surgery

TITANIUM ENDOSSEOUS IMPLANTS

Osseointegration:

- Direct adaptation of bone to dental implant.
- Histologic definition.
 - Evident at the light microscope level.

1989 implant success criteria

- Implant is immobile when tested clinically.
- No peri-implant radiolucency present.
- Mean vertical bone loss is less than 0.2 mm annually after the first year of placement.
- No persistent pain, discomfort, or infection.
- Implant design does not preclude placement of an esthetically acceptable crown or prosthesis.

Treatment planning:

- Physical evaluation of the oral hard and soft tissue
- Radiographic evaluation of quantity/quality of bone:
 - Panoramic radiograph
 - Computed tomography (CT) scan (DentaScan)
- Contraindications to implant therapy:
 - Uncontrolled diabetes
 - Immunocompromised patients
 - Anatomic considerations
 - Volume and height of bone
 - Bisphosphonate therapy
 - Bruxism
 - Tobacco use (relative contraindication)

Three stages of dental implant treatment:

1. Implant placement.
 - Facilitated by a surgical guide stent.
 - Proper angulation and parallelism.
 - Biologic width:
 - Dimension of healthy gingival tissues above alveolar bone.
 - Epithelial attachment (0.97 mm) plus connective tissue attachment (1.07 mm).
 - Average value = 2 mm.
 - A space of approximately 3 mm should be present between each implant and between implants and natural teeth.
2. Healing abutment placement.
3. Prosthetic restoration of implant.

EDENTULOUS MANDIBLE

- Tissue-born removable prosthesis
 - Placed over 1–5 implants.
 - Most often placed over 2–4 implants.
- Implant-born prosthesis
 - Five implants.
 - Placed in anterior mandible.
 - Placed anterior to mental foramen.

EDENTULOUS MAXILLA

- Parel's classification system
 - Class I: Patient missing maxillary teeth. Bone height retained.
 - Class II: Patient has lost teeth and some alveolar bone.
 - Class III: Patient has lost teeth and most of alveolar bone to basal level.
- Implant restoration of maxilla
 - Placement of implants at least 10 mm in length.
 - Placement of 4–8 endosseous implants.
 - Cross-arch stabilization of prosthesis.

Bone Augmentation Surgery

- Maxillary sinus grafts (sinus lift surgery)
 - Augmentation of the maxillary alveolar ridge.
- Bone grafting
 - Allograph
 - Transplanted from one individual to another genetically nonidentical individual (same species).
 - Autograph
 - Transplanted from one region to another (same individual).
 - Cortical bone grafts.
 - Cancellous bone grafts.
 - Jawbone (chin, maxillary tuberosity).
 - Iliac crest.
 - Tibia bone.
 - Xenograft
 - Transplanted from one species to another.
- Guided tissue regeneration
 - Membranes are used to hinder the migration of fibrous connective tissue while supporting the growth of bone.
- Alveolar distraction osteogenesis:
 - Alveolar “lengthening.”
 - That is, goal of 10–12 mm in the anterior mandible.
 - Bone fragments are moved “physiologically.”
 - Bone is formed in the distraction zone.
 - Latency period required prior to distraction.
 - 1 week
 - Allows for incisional healing.
 - 0.5 mm–1 mm increments of movement/day.
 - 1 mm movement not to be exceeded in 24 hours.
 - Bone fill can take 10–12 weeks to form.
 - Evaluated radiographically.



Augmentation of an atrophic mandible prior to implant placement is indicated in a patient with less than 8 mm of bone height.



Smokers, alcoholics, patients with uncontrolled diabetes or other uncontrolled systemic disease are poor candidates for bone-grafting procedures.



No treatment is needed for intruded primary teeth. Just await reeruption.



Always confirm with radiograph after repositioning a tooth/teeth.



Nonrigid splint is recommended for subluxation, luxation, and avulsion to avoid ankylosis.



Pulp necrosis is common with tooth luxation and avulsion.



Avulsed teeth can be preserved in saliva, fresh milk, or Hanks balanced salt solution. The tooth should be placed in the appropriate solution within 15–20 minutes, and reimplanted as soon as possible (best result within 30 minutes).

Trauma

DENTOALVEOLAR INJURIES

Always perform a thorough clinical examination (soft and hard tissue, percussion, mobility, and pulp testing/vitality) followed by radiographic evaluation (periapical/occlusal/panoramic).

Type of injuries:

- Crown fracture:
 - May involve enamel, dentin, or pulp.
 - Smooth off the sharp edges of enamel.
 - Seal the dentinal tubules with calcium hydroxide or glass ionomer cements.
 - If the pulp is exposed:
 - Permanent teeth:
 - Calcium hydroxide cap (best prognosis if immature apex).
 - Pulpectomy if insufficient tooth structure remaining.
 - Primary teeth: Pulpotomy
- Crown-root fracture:
 - If the pulp is exposed:
 - Permanent teeth:
 - $>1/3$ of root involved → Extraction
 - $<1/3$ of root involved → Pulpectomy
 - Primary teeth: Extraction
- Root fracture (divided in third—apical, midroot, coronal)
 - Coronal: If fracture communicate with gingival sulcus → Removal of coronal segment and perform pulpectomy.
 - Apical and midroot: Rigid splint for 2–3 months.
- Concussion (sensitivity without mobility or displacement):
 - No treatment needed.
- Subluxation (tooth mobility without tooth displacement):
 - Occlusal adjustment if needed.
 - Splint for 7–10 days to minimize pain if needed.
- Luxation (extrusion/intrusion/lateral/labial/lingual):
 - Reposition and splint for 14 days.
 - Occlusal adjustment if needed.
 - If alveolar process is fractured, splint for 4–6 weeks.
 - Intrusion: Await reeruption or may require orthodontic extrusion.
- Avulsion (complete extrusion):
 - Minimize contact with root (do not scrape the PDL or sterilize the tooth).
 - Irrigate with normal saline.
 - Replant and splint for 7–10 days (3–4 weeks for immature apex).
 - Adjust occlusion as needed.
 - Do not replant primary teeth.
- Alveolar process fracture:
 - Reduce dentoalveolar segment
 - Adjust occlusion as needed
 - Rigid splint (arch bar) for 4–6 weeks

Remember to follow up with frequent clinical and radiographic evaluation.

MAXILLOFACIAL FRACTURES

- Classification of fractures:
 - Simple (closed): No communication with the external environment
 - Compound (open, complex): Communication with external environment (via skin, mucosa, or periodontal ligament)
 - Comminuted: Fractured in multiple pieces
 - Greenstick: One side of the bone is broken and the other is bent
 - Pathologic: Occurred at a weakened site due to preexisting disease
- Treatment goals of maxillofacial fractures:
 - Control hemorrhage
 - Restore occlusion
 - Reduction of fractured segments
 - Stabilization of fractured segments
- Mandibular fractures (see Fig. 4–10):
 - Second most common fractured facial bone
 - 50% of mandibular fractures are multiple
- Classification:
 - Favorable: Not displaced by the forces of the muscles of mastication
 - Unfavorable: Displaced by the pull of the muscles of mastication
 - Horizontal (cephalad-caudal) vs. vertical (bucco-lingual)
- Signs and symptoms:
 - Malocclusion
 - Step defects (occlusal or inferior border of mandible)
 - Mobility of mandibular segments
 - Mucosal lacerations
 - Floor of mouth ecchymosis
 - Paresthesia of inferior alveolar nerve (CN V3)



When there is an associated lip laceration with a fractured tooth, always take a soft tissue radiograph to detect any broken fragments of tooth material.



Nasal bone fracture is the most common facial fracture.



Bilateral mandible fractures may result in posterior displacement of the tongue resulting in airway obstruction.



Fracture of the condyle will result in deviation on opening to the fractured side, due to trauma to the ipsilateral lateral pterygoid muscle—therefore, fracture of the right condyle will cause deviation of the mandible to the right upon opening.

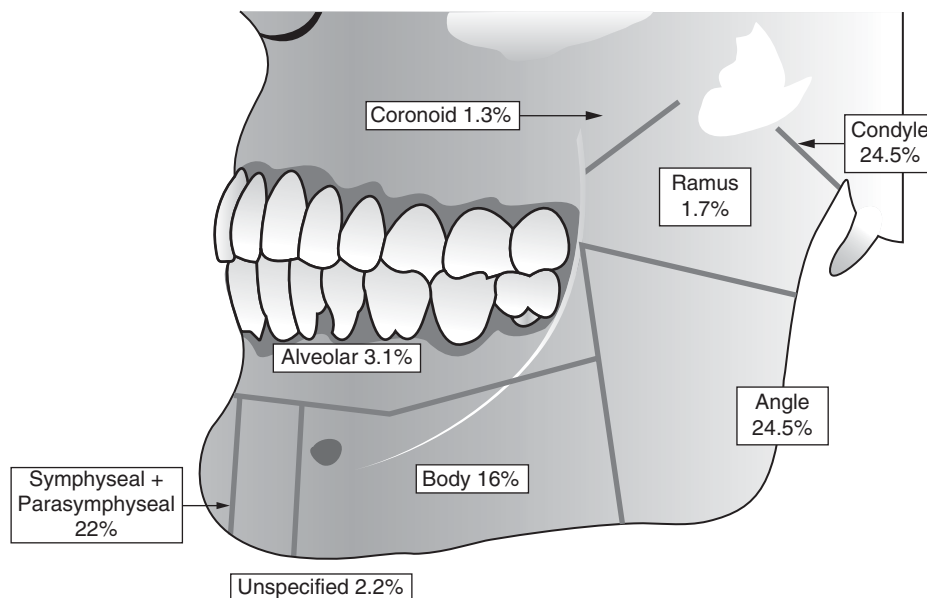


FIGURE 4–10. Anatomical distribution and frequency of mandible fractures.



Prolonged immobilization of condylar fractures will lead to fibrous or bony ankylosis of the temporomandibular joint (TMJ). Condylar fractures, which require closed or open reduction, should be immobilized for a maximum of 2 weeks, followed by mobilization with a regimen of passive jaw-opening exercises alternating with intermaxillary elastic fixation.



A “blow-out fracture” is an orbital floor fracture. Evaluate for entrapment of the extraocular muscles (inferior oblique) resulting in diplopia. These fractures may result in enophthalmos as a late complication.

- Imaging:
 - Panoramic (gold standard)
 - Mandibular film series (posteroanterior, reverse Towne’s, lateral oblique right and left)
 - Occlusal and periapical films
 - CT scan
- Treatment:
 - Closed reduction/maxillomandibular fixation (MMF)
 - Placement of arch bars, wires, rubber bands
 - External fixation
 - Open reduction and internal fixation
- Midface fractures (see Fig. 4–11)
- LeFort I (transverse maxillary):
 - Mobility of maxilla (usually displaced posteriorly and inferiorly) with intact nasofrontal complex
 - Malocclusion
 - Buccal vestibule ecchymosis (Guerin’s sign)
 - Epistaxis
- LeFort II (pyramidal):
 - Mobility of maxilla with mobile nasofrontal complex
 - Malocclusion (open bite or lengthening of face)
 - Periorbital edema and ecchymosis
 - Subconjunctival hemorrhage
 - Paresthesia of infraorbital nerve
 - Epistaxis

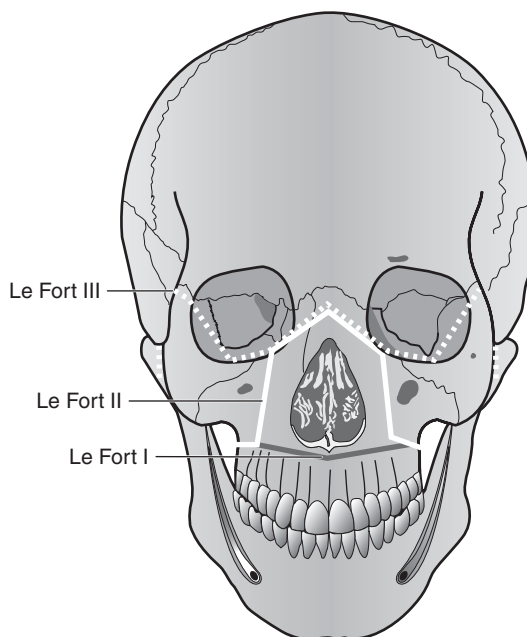


FIGURE 4–11. Le Fort fracture lines.

- Lefort III (craniofacial dysjunction)
 - Mobility of maxilla with mobile nasofrontal and malar complexes
 - Malocclusion
 - Periorbital edema and ecchymosis
 - Subconjunctival hemorrhage
 - Epistaxis
 - Rhinorrhea (cerebrospinal fluid [CSF] leak into nasal cavity)
- Zygomaticomaxillary complex fracture (ZMC):
 - “Cheek bone” fracture
 - Flattening of malar prominence
 - Periorbital edema and ecchymosis
 - Subconjunctival hemorrhage
 - Paresthesia of infraorbital nerve
 - Buccal vestibule ecchymosis
 - May have limited mouth opening if interfere with coronoid process
- Zygomatic arch fracture:
 - Isolated or in combination with ZMC fracture
 - Trismus if it interferes with the coronoid process
 - “W” deformity on submental vertex radiograph and CT scan (see Fig. 4–12)
- Imaging:
 - CT scan—Most valuable
 - Plain films (Water’s view, lateral skull, posteroanterior, and submentovertex)
- Treatment:
 - For displaced fractures which cause functional and/or esthetic deficits:
 - Direct exposure of all involved fractures
 - Reduction and anatomic realignment of the maxillary buttresses (nasomaxillary, zygomatic, pterygomaxillary)
 - Restoration of occlusion with MMF
 - Internal fixation using miniplate fixation



The zygoma articulates with four bones (frontal, temporal, maxillary, and sphenoid).



The submental vertex view is especially useful to diagnose an isolated zygomatic arch fracture.



For isolated zygomatic arch fractures a Gillies approach (deep layer of the superficial temporal fascia) can be used to insert a long elevator to “pop” the zygomatic arch back into position. Alternatively, an intraoral approach can be used with an incision in the posterior maxillary vestibule and insertion of a long elevator to reduce the zygomatic arch fracture.

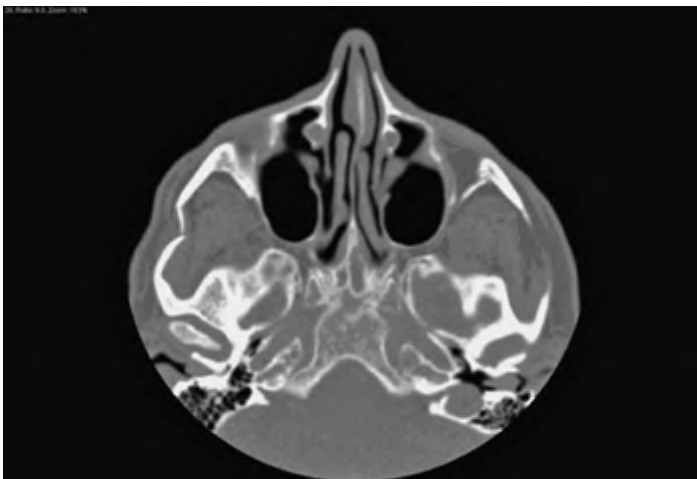


FIGURE 4–12. CT scan axial image showing right zygomatic arch fracture—“W” deformity.

Orthognathic Surgery

- For correction of skeletal malocclusion.
- Usually performed in conjunction with orthodontic treatment to reverse the dento-alveolar compensatory response to skeletal facial deformity.
- Cephalometric analysis and surgical models play an important role in preoperative work-up.

Maxillary Surgery

- Lefort I osteotomy

Mandibular Surgery

- Bilateral sagittal split ramisectomy (BSSR)
- Intraoral vertical ramus osteotomy (IVRO)
- Genioplasty



Cleft lip rule of 10's: Surgery is performed when the child is at least 10 weeks of age, weighs at least 10 lb, and has at least 10 g/dL hemoglobin.

Cleft Lip and Palate Surgery

- Orofacial clefts (see Fig. 4–13):
 - Occurrence: 1 in 600–1000 live births in the United States.
 - Ratio 3:2, male: female.
 - Cleft lip is more common in males.
 - Cleft palate is more common in females.
 - Most commonly found in Asians, least commonly found in African Americans.
 - Embryological defect occurs between the fifth and tenth week of fetal life.
 - Cleft lip: Lack of fusion between lateral and medial nasal processes.
 - Cleft palate: Lack of fusion between palatal shelves.

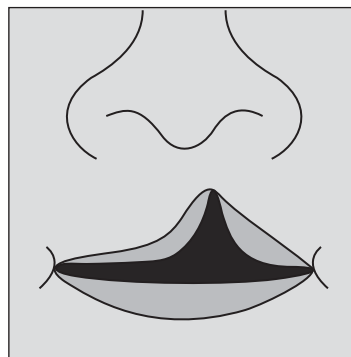


FIGURE 4–13A. Unilateral incomplete cleft lip (left side).

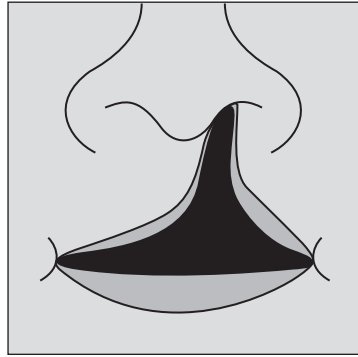


FIGURE 4-13 B. Unilateral complete cleft lip (left side).

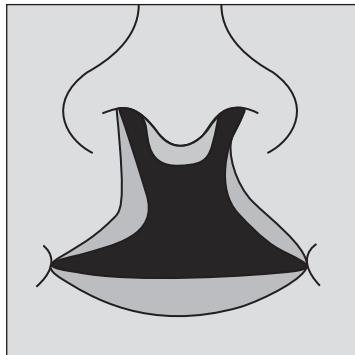


FIGURE 4-13 C. Bilateral complete cleft lip.

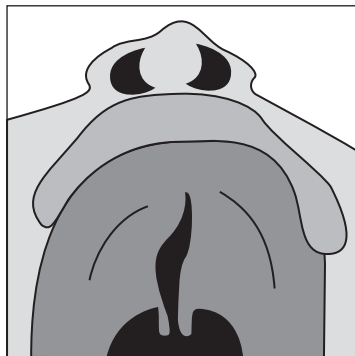


FIGURE 4-13 D. Cleft palate.

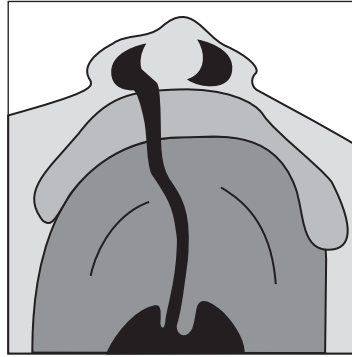


FIGURE 4-13E. Unilateral cleft lip and palate.

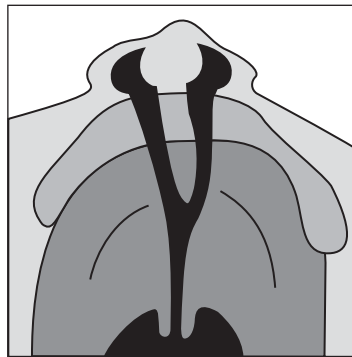


FIGURE 4-13F. Bilateral cleft lip and palate.

- Problems associated with orofacial clefts:
 - Feeding
 - Speech
 - Ear problems (otitis)
 - Malocclusion
 - Nasal deformities
 - Psychosocial

► FACIAL PAIN

Temporomandibular Joint (TMJ)

- Classification:
 - Ginglymoarthrodial joint
 - Translational (gliding) movement
 - Rotational (hinging) movement
 - Synovial joint

Anatomy

- TMJ (see Fig. 4–14):
 - Articulation between the condyle of the mandible and the squamous portion of the temporal bone (TMJ fossa).
 - Articular disc lies between condyle and fossa.
- Condyles:
 - Elliptically shaped.
 - Long axis-oriented mediolaterally.
- Articular surface of temporal bone
 - Functional aspect of TMJ.
 - Dense fibrous connective tissue.
 - Concave: Articular fossa (glenoid fossa, mandibular fossa)
 - Convex: Articular eminence (tubercle)
- Articular disc:
 - Dense fibrocartilaginous connective tissue.
 - Avascular and aneural.
 - Nutrition of chondrocytes with the movement of synovial fluid is essential for maintenance of structure and function.
 - Biconcave structure is a three-dimensional space filler between the two convex surfaces of the condyle and articular eminence.
 - Separates joint into inferior and superior joint spaces.
 - Varies in thickness:
 - Intermediate zone—thin (center of disc)
 - Anterior and posterior bands—thick
 - Posterior band is thicker and is attached to retrodiscal tissues (bilaminar zone, posterior attachment).



Joint noise is not necessarily representative of a pathologic condition. Clicking or crepitus without pain and dysfunction should be followed but do not require any treatment.



Synovial inflammation can cause alterations in the occlusion, with difficulty obtaining a comfortable posterior occlusion. Occlusal adjustments should not be performed especially in the presence of synovial inflammation. Severe synovial inflammation is associated with pain with lateral excursion movements in the ipsilateral joint.

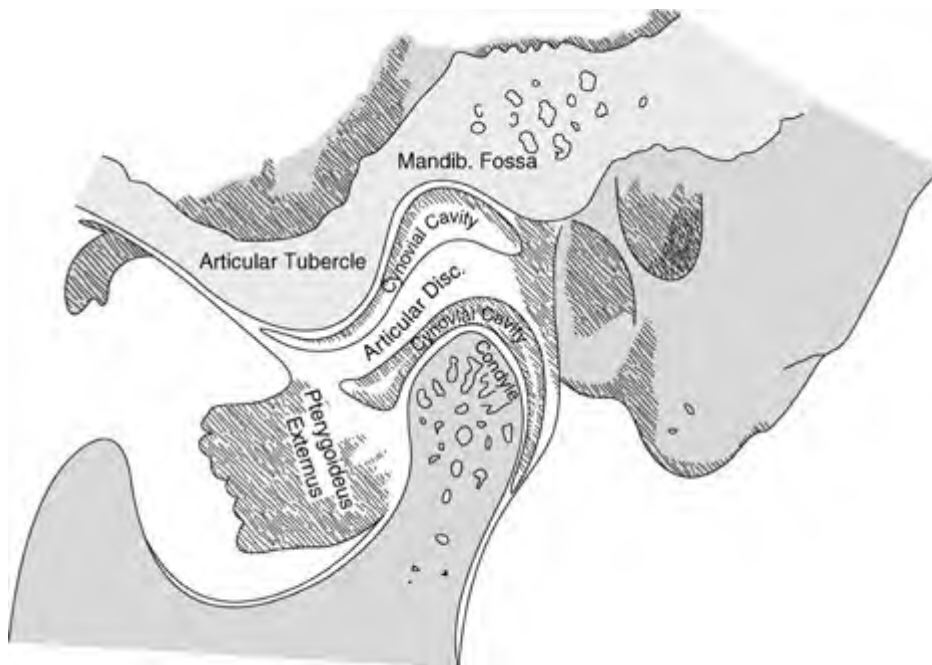


FIGURE 4–14. Sagittal section of the articulation of the mandible.

- Anterior band is attached to the capsular ligament, the lateral pterygoid muscle, and the condyle.
- Retrodiscal tissues:
 - Loose connective tissues.
 - Vascular and innervated.

Muscular Attachments of Mandible

- Muscles of mastication:

Muscle	Origin	Insertion	Function
Temporalis	Floor of temporal fossa, deep surface of temporal fascia	Coronoid process, anterior border of mandibular ramus	Elevates and retrudes mandible
Masseter	<i>Superficial portion</i> —anterior 2/3 of lower border of zygomatic arch <i>Deep portion</i> —medial surface of zygomatic arch	Lateral surface of ramus, coronoid process, and angle of mandible	Elevates, protrudes, and retrudes mandible
Lateral Pterygoid	<i>Superior head</i> —infratemporal surface of sphenoid greater wing <i>Inferior head</i> —lateral surface of lateral pterygoid plate	Anterior portion of condylar neck and TMJ capsule	Protrusion of mandible; lateral movements of mandible
Medial Pterygoid	<i>Deep head</i> —medial surface of lateral pterygoid plate; pyramidal process of palatine bone <i>Superficial head</i> —maxillary tuberosity	Medial surface of ramus, inferior to mandibular foramen	Protrudes and elevates mandible; lateral movements of mandible

- Suprahyoid muscles:

Muscle	Origin	Insertion	Function
Geniohyoid	Inferior genial tubercle on inner surface of mandibular symphysis	Anterior surface of hyoid bone body	Elevates tongue and hyoid
Mylohyoid	Line from last molar to mandibular symphysis	Median raphe from chin to hyoid bone and hyoid bone	Elevates base of tongue and hyoid bone. Raises floor of mouth. Depresses mandible when hyoid bone is fixed
Digastric	<i>Posterior belly</i> —mastoid notch (temporal bone) <i>Anterior belly</i> —digastric fossa (mandible)	Intermediate tendon attached to hyoid bone by fibrous loop	Elevates the base of tongue and hyoid bone
Stylohyoid	Posterior border of the styloid process	Body of hyoid bone at junction with greater horn	Elevates base of tongue and hyoid bone

- Accessory muscles to the muscles of mastication:
 - Platysma
 - Buccinator
 - Posterior neck musculature:
 - Sternocleidomastoid
 - Trapezius
 - Intrinsic neck muscles
- Centric relation position
 - Anatomic relationship of the TMJ joint

Examination of the TMJ:

- Evaluate for tenderness to palpation.
- Palpate the lateral aspect of the joint while the patient opens and closes mouth.
- Palpate the anterior wall of the external auditory meatus to palpate the posterior aspect of the mandibular condyle.
- Inflammation of the synovial tissues and capsule is characterized by pain with palpation.
- Measure mandibular range of motion including vertical, lateral, and protrusive movements.
- Palpate and auscultate both joints to assess clicking and crepitus.

TMJ Disorders (TMD)

MUSCULAR DISORDERS

- Myofascial pain dysfunction (MPD) syndrome
 - Masticatory myalgia.
 - Most common form of TMJ pain.
 - Muscles of mastication are primarily affected.
 - Stress-related disorder:
 - Increased stress causes increased muscle tension and teeth clenching/bruxism.
 - Characterized by muscle spasm, pain, and dysfunction.

NERVE DISORDERS

- Trigeminal neuralgia (tic douloureux)
 - Usually described as stabbing, burning, shocking pain lasting seconds to minutes.
 - Provoked by wind, tactile, or thermal stimulation.
 - Affects one or more divisions of a unilateral trigeminal nerve.
 - Usually unilateral.
 - No motor or sensory nerve deficit.
 - Treatment: Antiepileptics (carbamazepine), gabapentin.
 - Surgery for patients refractory to medical therapy:
 - Microvascular decompression.
 - Gamma knife.
- Conservative (nonsurgical) treatment of MPD:
 - Fabrication of an acrylic night guard (occlusal separator appliance).
 - Soft diet.
 - Gentle passive range of motion exercises.
 - Moist heat, massage, and ice.
- Reduction of joint overloading (softer diet, elimination of gum chewing).
- NSAIDS and application of moist heat during acute exacerbations.

INTERNAL TMJ DISORDERS

- Disc displacement disorders:
 - Often associated with synovial inflammation.
 - Disc displacement is usually the end result of inflammation and chronic joint overloading.
 - Abnormal relationship between the disc and the condyle.
- Internal derangement:
 - Abnormal relationship of the articular disc to the mandibular condyle, fossa, and articular eminence.
 - Posterior band of the articular disc is anteriorly displaced in front of the condyle.
 - As the articular disc translates anteriorly, the posterior band remains in front of the condyle.
 - The retrodiscal tissue (bilaminar zone) is extremely inflamed and functional loading causes further significant inflammation.
 - Inflamed synovial tissues are not able to produce an adequate synovial fluid, resulting in poor lubrication and reduced mobility.
- Anterior displacement with reduction:
 - Displaced posterior band will return to its anatomical position.
 - When the articular disc reduces, the patient often experiences “clicking” or “popping.”
- Anterior displacement without reduction:
 - Condyle unable to translate fully to its most anterior position.
 - Patient unable to open maximally.
 - Deviation of the mandible to the affected side.
 - No clicking occurs in these patients.
 - Significant synovial inflammation and adhesions are associated with this condition.
- Chronic recurrent dislocation:
 - Secondary to mandibular hypermobility.
 - Mandibular condyle dislocates anterior to the articular eminence
 - Secondary severe spasm of the muscles of mastication cause additional severe pain and often makes reduction of dislocation difficult.
 - Treatment involves manual manipulation of the posterior mandible inferiorly to get the condyle below the articular eminence and then posteriorly to position into the glenoid fossa.
 - Local anesthesia into the muscles of mastication and/or intravenous sedation may be necessary to overcome the severe masticatory muscle spasm.
 - Following reduction of the dislocation, the clinician may decide to prevent dislocation by using temporary elastic maxillomandibular fixation.

DEGENERATIVE JOINT DISEASE (DJD)

- Osteoarthritis
- Systemic arthritic conditions:
 - Rheumatoid arthritis
 - Systemic lupus erythematosus (SLE)
 - Psoriatic arthritis

- TMJ ankylosis
 - Bone or fibrous tissue fusion of condyle, disk, and TMJ fossa.
 - Most commonly caused by trauma.
 - Most common complication of rheumatoid arthritis.

TMD TREATMENT

- **Reversible therapy**
 - Patient education
 - Medication (NSAIDs and muscle relaxants)
 - Physical therapy
 - Splint therapy—designed to unload the joint:
 - Equal occlusion throughout the arch
 - No anterior or posterior positioning
 - Passive motion exercises (gentle range of motion movements of the mandible with fingers or device, not activated by the muscles of mastication)
- **Irreversible therapy**
 - Occlusal adjustments (AVOID)
 - Prosthetic restoration (AVOID)
 - Orthodontic treatment (AVOID)
 - Orthognathic surgery (AVOID)
 - TMJ surgery
- Surgical approach to the TMJ—indications:
 - Conservative treatment fails to reduce symptoms.
 - Symptoms must be caused by intra-articular pathology.
 - Symptoms have significant affect on quality of life.
- Arthrocentesis:
 - Placement of two needles into superior joint space.
 - Lavage of joint space to reduce inflammatory mediators.
 - Indicated with recent onset of unilateral failure of translation due to internal derangement.
- Arthroscopic surgery:
 - Access into the superior joint space to directly visualize intra-articular pathology—adhesions, synovitis, osteoarthritis, disc displacement, disc perforation.
 - Operative arthroscopy is used to treat the pathology that is seen.
 - Lysis of adhesions.
 - Direct steroid injection into synovitis.
 - Motorized shaving of osteoarthritic fibrillation tissue and around the perforated disc.
- Open surgical procedures:
 - Indicated primarily with ankylosis and joint pathology when there is no joint space.
 - Arthroplasty: Disk repair or removal.
 - Condylotomy: Subcondylar osteotomy acts as a stress breaker.
 - Total joint replacement.
- Preauricular incision
 - The best surgical approach to expose the TMJ.
 - Perpendicular incision.
 - Anterior to the external ear.



DJD is characterized by loss of the integrity of the articular cartilaginous surfaces. Joint overloading and joint immobilization are major factors that cause breakdown of the articular cartilage matrix.



Routine radiographs will appear normal in early osteoarthritis, but it is readily apparent on arthroscopic examination. Advanced disease will ultimately result in changes to the subchondral bone and is associated with irregular, perforated, and severely damaged discs as well as articular surface abnormalities (flattening, erosions, osteophytes).



Systemic conditions tend to affect TMJs bilaterally.



The most commonly used medications to treat TMD include NSAIDs, stronger analgesics, muscle relaxants, and tricyclic antidepressants.



There are no studies that demonstrate that the occlusion is the etiology of TMJ disorders.

- Parallel to the superficial temporal artery.
- Incision extends from one inch above the zygomatic arch to the lower aspect of the ear.
- Approaches the condyle posteriorly.
- Submandibular approach (risdon incision):
 - Standard surgical approach to the ramus of the mandible and neck of the condyle.
 - Not the best approach for procedures within the joint space itself.

► LESIONS

- Biopsy
 - Incisional
 - Removal of a representative portion of a lesion.
 - Excisional biopsy
 - Removal of entire lesion at time surgical diagnostic procedure is performed.
 - Usually an elliptical shaped incision is used.
 - Aspiration biopsy:
 - Use of a needle and syringe to sample the contents of a lesion. (FNA = fine needle aspiration).
- Tori (exostosis)
 - Bony growths most often found on palate or lingual aspect of mandible.
 - May interfere with speech.
 - Can become ulcerated following oral trauma.
 - May need to be removed prior to fabrication of complete or partial removal denture.
 - Postoperative hematoma formation is prevented by using an acrylic splint, temporary denture, pressure dressing, or insertion of a drain.
- Sialolithiasis (salivary gland stones)
 - Most often affects submandibular gland (85%).
 - Causes pain and swelling.
 - Swelling worsens when salivary flow is stimulated.
 - Gland may become infected, causing purulent discharge, erythema, floor of mouth edema, and lymphadenopathy.
 - Treatment: stone removal, sialodochoplasty, or gland removal.

► INFECTIONS

Microbiology

Common bacteria found in oral infections:

- Aerobic (25%):
 - Gram-positive cocci:
 - *Streptococcus* spp. (mostly α -hemolytic)
 - *Staphylococcus* spp.
 - Gram-negative cocci (*Neisseria* spp.)
 - Gram-positive rods (*Corynebacterium* spp.)
 - Gram-negative rods (*Haemophilus*, *Eikenella*, and *Enterobacteriaceae* spp.)
- Anaerobic (75%):
 - Gram-positive cocci:
 - *Streptococcus* spp.
 - *Peptostreptococcus* spp.

- Gram-negative cocci (*Veillonella* spp.)
- Gram-positive rods:
 - *Eubacterium* spp.
 - *Lactobacillus* spp.
 - *Actinomyces* spp.
 - *Clostridia* spp.
- Gram-negative rods:
 - *Bacteroides* spp.
 - *Fusobacterium* spp.

Progression of odontogenic infection:

1. Usually start with highly virulent aerobic bacteria (usually strep) → Cellulitis (acute) (see Table 4–6)
2. Progressed to mixed aerobic and anaerobic infections
3. Then mainly anaerobic bacteria (fusobacterium and bacteroides) → Abscess (chronic) (see Table 4–6)

The spread of odontogenic infection depends on:

- Thickness of bone next to infected tooth
- Position of muscle attachment in relation to root tip
- Virulence of the bacteria
- Patient's host defense
- Primary fascial spaces:
 - Maxillary:
 - Buccal
 - Canine
 - Infratemporal
 - Mandibular:
 - Buccal
 - Submental
 - Submandibular
 - Sublingual



Most odontogenic infections are from indigenous bacteria and polymicrobial in nature, comprising of both aerobic and anaerobic bacteria.



Infection spreads down the path of least resistance.

TABLE 4 – 6 . Cellulitis vs. Abscess

CHARACTERISTIC	CELLULITIS	ABSCESS
Duration	Acute	Chronic
Pain	Severe and generalized	Localized
Size	Large	Small
Localization	Diffuse border	Well circumscribed
Palpation	Doughy to indurated	Fluctuant
Presence of pus	No	Yes
Degree of seriousness	Greater	Less
Bacteria	Aerobic	Anaerobic



Abscesses should be drained to establish dependent drainage (from the most gravity-dependent point).



"The solution to pollution is dilution." (Irrigate copiously with normal saline solution.)



No antibiotic is necessary for treatment of a dry socket.

- Secondary fascial spaces
 - Masseteric/masticator
 - Pterygomandibular
 - Superficial and deep temporal
 - Lateral pharyngeal
 - Retropharyngeal
 - Prevertebral
- Ludwig's angina
 - Bilateral infection/induration of the following spaces:
 - Submandibular
 - Sublingual
 - Submental
 - Management of the airway is a primary concern in this type of infection.
- Principles of management of odontogenic infections:
 - Determine severity (anatomic location, rate of progression, airway compromise (trismus, dysphagia, dyspnea, dysphonia)
 - Evaluate host defenses
 - Evaluate need for referral to oral surgeons
 - Treat surgically (endodontic treatment, extraction, incision, and drainage)
 - Culture and sensitivity (especially with severe or chronic infection, and in immunocompromised patient)
 - Appropriate antibiotics
 - Support medically (analgesic, fluids, nutrition)
 - Frequent evaluation
- Incision and drainage:
 - Treatment of infection
 - Changing the environment to one less optimal for bacterial growth
 - Drain placement
 - Irrigation
- Indication for antibiotics
 - Rapidly progressing infection
 - Diffuse edema
 - Compromised host defenses (diabetes, cancer, steroids, HIV/AIDS, alcoholic, malnutrition, chronic renal disease)
 - Involvement of fascial spaces
 - Severe pericoronitis
 - Osteomyelitis of the jaw
- Empiric antibiotics for odontogenic infections
 - Penicillin (first choice)
 - Amoxicillin
 - Augmentin (amoxicillin-clavulanate)
 - Clindamycin (first choice if allergic to penicillin)
 - Azithromycin
 - Clarithromycin
 - Flagyl
- Necrotizing fasciitis (cervicofacial)
 - Rapidly progressing infection of the skin and fascia
 - High mortality rate (30–50%)
 - Etiology: Group A Strep, mixed aerobic–anaerobic bacteria, or *C. perfringens*
 - Treatment: Surgical debridement and antibiotic therapy

- Cavernous sinus thrombosis:
 - The head and neck is drained by valveless veins, which permit retrograde flow from the face to the sinus.
 - Cranial nerves involved: III, IV, V1 (ophthalmic), and VI.
 - Vascular congestion (sclera, retina), periorbital edema, proptosis, ptosis, dilated pupils, absent corneal reflex, and thrombosis of retinal veins.
- Sinusitis:
 - Infection of the sinuses (collection of pus), most commonly the maxillary sinuses
 - Acute (symptoms <1 month)
 - Etiology:
 - *S. pneumoniae* (30–50%)
 - *H. influenzae* (20–40%)
 - *M. catarrhalis*
 - Viruses
 - Chronic (symptoms >3 months)
 - Etiology:
 - Obstruction of sinus drainage
 - Chronic low-grade anaerobic infection
 - Diabetics may develop mucormycosis (fungus)
 - Treatment:
 - Amoxicillin, augmentin
 - Symptomatic therapy (nasal decongestant, antihistamines, pain medications)
 - Surgery (reestablish drainage and remove infected tooth/source of infection)
 - Complications: Orbital cellulitis, cavernous sinus thrombosis, meningitis, osteomyelitis, intracranial abscess
- Infections from animal bites
 - Mainly caused by *Pasteurella multocida*.
 - Treatment: Ampicillin or amoxicillin.



Beware of antibiotic-associated colitis (Clostridium difficile) with amoxicillin, clindamycin and cephalosporins. Treatment is metronidazole or oral vancomycin.

Orthodontics

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Classification of Occlusion (See Fig. 5–1)

- Class I:
 - 70% of population.
 - The mesiobuccal cusp of the maxillary first molar lines up with the buccal groove of the mandibular first molar.
 - The maxillary central incisors overlap the mandibular incisors.
 - The maxillary canine lies between the mandibular canine and first premolar.
- Class II:
 - Can also be referred to as a **distoclusion**, **retrognathism**, or overbite.
 - 25% of population.
 - The mesiobuccal cusp of the maxillary first molar falls between the mandibular first molar and the second premolar.
 - The lower jaw and chin may also appear small and withdrawn.
 - Maxillary canine is mesial to mandibular canine.
 - **Class II, Division 1:** Maxillary incisors (centrals and laterals) in extreme labioversion (protruded).
 - **Class II, Division 2:** Maxillary centrals tipped palatally and in a retruded position (linguoversion). The maxillary laterals are typically tipped labially and mesially.
 - **Subdivision:** The malocclusion occurs on one side of the dental arch only.
 - Example: Class II, Division 1 subdivision right—The right molar is in a class II relationship while the left molar is in a class I relationship.
- Class III:
 - Less than 5% of the population.
 - The mesiobuccal cusp of the maxillary first molar falls between the mandibular first molar and the second molar.
 - The chin may be in a protrusive position.
 - The maxillary canine is distal to the mandibular canine.
 - Can also be referred to as a **mesioclusion**, **prognathism**, or underbite.

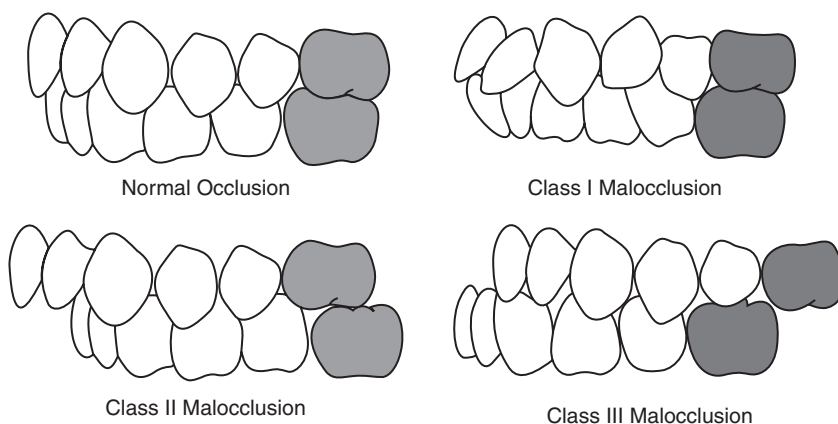


FIGURE 5–1. Classification of occlusion.

- Class III malocclusions are those in which the body of the mandible and its superimposed dental arch are in a mesial relationship to the skull base and maxilla. The maxillary first molar therefore occludes distal to the mandibular first molar, while the maxillary canine is in an exaggerated distal relationship to the mandibular canine.
- The overjet in a class I occlusion is 1–2 mm. In a class III malocclusion, the overjet is 0 mm (edge-to-edge bite) or negative. (The mandibular incisors are forward relative to the maxillary incisors.)
- **Pseudo Class III malocclusion:**
 - Describes a situation in which the patient adopts a jaw position upon closure which is forward to normal.
 - Typically, the pseudo class III patient presents with an edge-to-edge bite.
 - In order to avoid interference and achieve maximal intercuspation, the patient slides his/her jaw forward.
 - This patient has the ability to bring the mandible back **without strain** so that the mandibular incisors touch the maxillary incisors.
 - This type is therefore a milder form of the class III malocclusion and more amenable to conservative orthodontic treatment than the “true” class III malocclusion which often requires surgical correction.

Signs of Incipient Malocclusion

- The lack of interdental spacing in the primary dentition.
 - The significance of the lack of spacing relates to the increased mesio-distal width of the permanent teeth.
- The crowding of the permanent incisors in the mixed dentition.
 - Arch perimeter increases slightly after the eruption of the incisors, but after this stage of dental development, arch length reduces as a result of the loss of E space. Hence, crowding of the permanent incisors in the early mixed dentition typically results in crowding in the permanent dentition.
- The premature loss of the primary canines, particularly in the mandibular arch.
 - This phenomenon is indicative of insufficient arch size in the anterior region. During eruption of the lateral incisors, the crown of the laterals impinge on the roots of the primary canines and causes them to resorb. When the canine is shed, the midline will shift in the direction of the lost tooth. You will have lateral and lingual migration of the mandibular incisors.

Speech Difficulties Related to Malocclusion

Speech Sound	Related Malocclusion
s, z	Anterior open bite, large gap between incisors
t, d	Irregular incisors (especially lingual position of maxillary incisors)
f, v	Skeletal class III
th, sh, ch	Anterior open bite



An anterior crossbite of multiple teeth in the primary dentition usually indicates a skeletal growth problem.

Molar Uprighting

- Long-term loss of a mandibular permanent first molar:
 - Causes tipping, migration, and rotation of adjacent teeth into edentulous space.
 - Common reason why molar uprighing of second molar is required.
- Reasons why normal angulation of a molar is desirable:
 - Improves the direction and distribution of occlusal forces.
 - Decreases the amount of tooth reduction required for parallelism of the abutments.
 - Decreases the possibility of endodontic, periodontic, or more complex prosthodontic procedures.
 - Increases the durability of the restorations, due to better force distribution.
 - Improves the periodontal environment by eliminating plaque-retentive areas.
 - Improves the alveolar contour.
 - Improves crown-to-root ratio.
- **Best treatment method:** Tipping the crown of second molar distally and opening up space for a pontic to replace missing first molar.
 - Upright with fixed edgewise orthodontic appliance.
 - Bracket slot size: 0.022 in. or (alternatively, 0.018 in.).
 - Tipped second molar should be banded because of the considerable posterior masticatory forces produced can easily shear off bonded brackets.
 - Time frame: 6–12 months.
- Considerations:
 - Severely lingually tipped mandibular molar is more difficult to control and upright properly.
 - Stabilization should last until the **lamina dura and PDL** reorganize.
 - 2 months for simple uprighing.
 - 6 months for uprighing, osseous surgery, grafts, and the like.
 - Retention can be provided by an appliance or by a well-fitting provisional restoration.
 - Slow progress in molar uprighing in an adult patient is most likely due to **occlusal interferences**.
- Conditions that may complicate molar uprighing:
 - **High mandibular plane:**
 - If unsuccessful, it can lead to an increased open bite and loss of anterior guidance.
 - Open bite.
 - Presence of periodontal disease.
 - Poor crown-to-root ration and/or short roots.
 - Presence of root resorption.
 - Significant centric relation-to-maximum intercuspation discrepancy.
 - Severe lingual inclination of the tooth in addition to mesial tipping.
 - Occlusal plane disharmony (i.e., extruded maxillary and mandibular molars).
 - Severe skeletal discrepancies.

Impacted Teeth

- Most common: **Maxillary canine**
- May cause:
 - Damage to the roots of adjacent teeth.
 - Create severe orthodontic problem.
 - Create future prosthodontic problems to restore the missing tooth.



FIGURE 5-2. Exposure of maxillary canine.

(Reproduced, with permission, from Profitt WR, Fields HW, Sarver DM. Contemporary Orthodontics, 4th ed. Philadelphia, PA: Elsevier, 2007.)

- **Diagnosis:**
 - Can be first observed on an X-ray.
 - Association of impacted canines with missing lateral incisors or shortened roots of lateral incisors.
 - Distal aspect of the root of lateral incisors guides the eruption of canines.
- Older patients have an increased risk of having the impacted tooth becoming **ankylosed**.
- **Treatment:** Can be brought to the arch through orthodontic traction after being surgically exposed (see Fig. 5-2).
- Treatment planning considerations:
 - The overretained primary tooth should be considered for extraction based on its position and potential for eruption.
 - The prognosis should be based on the extent of displacement and the surgical trauma required for exposure.
 - During surgical exposure, flaps should be reflected so that the tooth is ultimately pulled into the arch through **keratinized tissue**, not through alveolar mucosa.
 - **Adequate space** should be provided in the arch **before** attempting to pull the impacted tooth into position.

► CEPHALOMETRICS

Radiographic Cephalometry

- Method of making indirect skull measurements utilizing X-rays and radiographic film.
- Linear and angular measurements are obtained utilizing known anatomical landmarks in the lateral head radiography of the patient.

- These measurements are then compared with those considered within normal limits and in that way enable the orthodontist to assess aberration in the dentition and jaw structures which result in malocclusion.
- The etiology of a particular malocclusion may have a dental component or a skeletal component or both.
- Application of cephalometric films:
 - To clarify the anatomic basis for a malocclusion and serve as a critical tool in orthodontic diagnosis.
 - To analyze treatment results. Cephalometric films taken before, during, and after treatment (serial cephalometrics) can be superimposed to study changes in jaw and tooth positions that have occurred due to orthodontic therapy.
 - To show the amount and direction of craniofacial growth.

Landmarks (See Fig. 5–3)

- **Bolton (Bo):** The highest point in the concavity behind the occipital condyle.
- **Basion (Ba):** The most forward and highest point of the anterior margin of foramen magnum.
- **Articulare (Ar):** The point of intersection of the contour of the posterior cranial base and the posterior contour of the condylar process.
- **Porion (Po):** Outer upper margin of the external auditory canal.
- **Sphenooccipital synchondrosis (SO):** Junction between the occipital and basisphenoid bones.
- **Sella (S):** The midpoint of sella turcica as determined by inspection.
- **Pterygomaxillary fissure (Ptm):** Point at base of fissure where anterior and posterior walls meet.

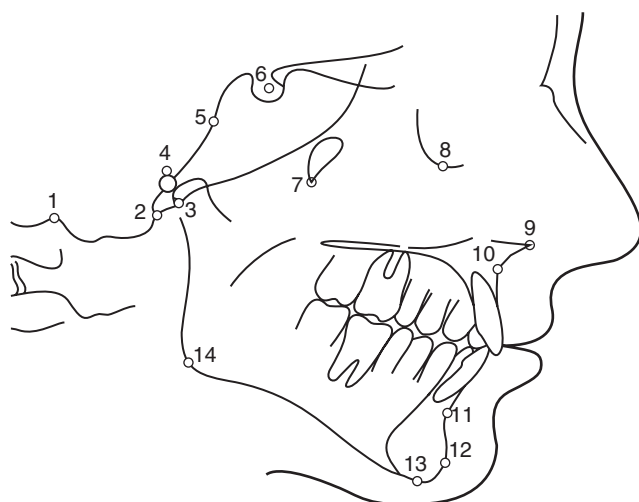


FIGURE 5–3. Lateral cephalometric tracing.

1 Bo; 2 Ba; 3 Ar; 4 Po; 5 SO; 6 S; 7 Ptm; 8 Or; 9 ANS; 10 Point A; 11 Point B; 12 Pog; 13 Me; 14 Go.



The Frankfort-Horizontal

Plane is constructed by drawing line connecting porion and orbital. This has been adopted as the best horizontal orientation from which to assess the lateral representation of the skull.

- **Orbitale (Or):** Lowest point on the inferior margin of the orbit.
- **Anterior nasal spine (ANS):** Tip of anterior nasal spine.
- **Point A (Subspinale):** Innermost point on contour of premaxilla between anterior nasal spine and incisor tooth.
- **Point B (Supramentale):** Innermost point on contour of mandible between incisor tooth and bony chin.
- **Pogonion (Pog):** Most anterior point of the contour of chin.
- **Menton (Me):** Most inferior point on the mandibular symphysis, the button of the chin.
- **Gonion (Go):** Lowest most posterior point on the mandible with the teeth in occlusion.

Mandibular Plane

- The mandibular plane angle can be visualized clinically by placing a mirror handle or other flat-edge instrument along the border of the mandible.
- Cephalometrically, the mandibular plane is the line connecting points gonion and menton (or gnathion).
- The mandibular plane angle is measured at the intersection formed by the mandibular plane and the sella-nasion line.
- A steep mandibular plane angle correlates with a long anterior vertical dimension and an anterior open bite malocclusion.
- A flat mandibular plane angle correlates with a short anterior facial vertical dimension and a deep bite malocclusion.

SNA Angle, SNB Angle, and ANB Angle

- An SNA angle of greater than 84° in the Caucasian population indicates maxillary prognathism.
- An SNB angle of less than 78° indicates mandibular retrognathism.
- An ANB angle of $2-4^\circ$ indicates a class I skeletal pattern.

► REMOVABLE APPLIANCES

Indications for Removable Appliances

- Limited tipping movements.
- Retention after comprehensive treatment.
- Growth modification during the mixed dentition.

Major Components of a Removable Appliance

- **Retentive component:** Examples include Adams clasp, ball clasp, C clasp, and arrow clasp.
- **A framework or baseplate:** This is typically made of acrylic and provides anchorage.
- **Active component or tooth moving component:** This consists of springs, jacks screws, or elastics.
- **Anchorage component:** This resists force of active component (e.g., acrylic base plate or labial bow or soldered arm).



The most common type of retainer is the Hawley retainer. (Other types of removable retainers include the Positioner and Essex retainer.)

Advantages and Disadvantages of Removable Appliances vs. the Fixed Appliances

- Advantages:
 - Improved hygiene.
 - Patient comfort increased.
 - Lab fabrication decreases chair time.
- Disadvantages:
 - Results heavily dependent on patient compliance.
 - Cannot achieve two-point tooth contact, thereby making bodily tooth movement impossible.
 - Removable appliances can only accomplish tooth tipping.

Headgear

- Headgear is typically used in skeletal class II growing patients to hold the growth of the maxilla back and to allow the mandible to catch up.
- Headgear needs to be worn 10–14 hours/day in order to be effective.
- Treatment length is typically 6–18 months, depending on the severity of the malocclusion.
- Headgear can also be used in adults for the maintenance of anchorage.
- Extraoral anchorage with the use of headgear allows the orthodontist to keep the posterior segment back without adversely disturbing the opposite arch.
- **High-pull headgear** consists of a head cap connected to a face bow. This appliance places a distal and intrusive force on the maxillary molars and maxilla.
- **Cervical-pull headgear** consists of a neck strap connected to a face bow. This appliance produces a distal and extrusive force against the maxillary teeth and maxilla.
- **Straight-pull headgear** is similar to the cervical-pull headgear. However, this appliance places a force in a straight distal direction.
- **Reverse-pull headgear** has an extraoral component that is supported by the chin and forehead. It is used in skeletal class III malocclusions to protract the maxilla.

► FUNCTIONAL APPLIANCES

- These appliances are designed to modify growth during mixed dentition.
- Functional appliances have a dental as well as a skeletal effect.
- Functional appliances can be categorized as tissue borne or tooth borne.
 - **Tooth-borne appliance:**
 - **Bionator:** Advances the mandible to an edge-to-edge position to stimulate mandibular growth for correction of class II malocclusion (see Fig. 5–4).
 - **Herbst:** Maxillary and mandibular framework splinted together via pin and tube device that holds the mandible forward (see Fig. 5–5).
 - **Tissue-borne appliance:**
 - The **Frankel functional appliance** is the only tissue-borne appliance.
 - It alters both mandibular posture and contour of facial soft tissue (see Fig. 5–6).

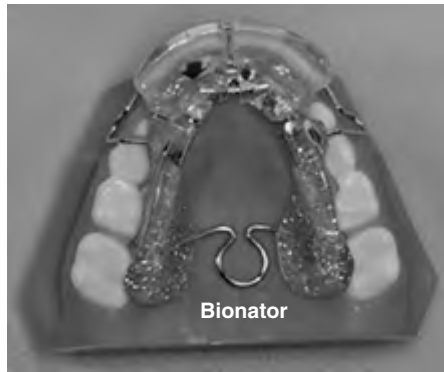


FIGURE 5-4. Bionator.

(Courtesy of Dockstader Dental Lab, Fresno, CA, www.dockstader.com.)



FIGURE 5-5. Herbst appliance.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)



FIGURE 5-6. Frankel appliance.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)

► FIXED APPLIANCES

Edgewise Appliance

- Horizontally positioned slot. (Earlier fixed appliances such as the **Begg appliance** used a vertically positioned slot.)
- Siamese twin bracket: Double wings for increased rotational and tip control of roots.

Straight-Wire Appliance

- A variation of the edgewise appliance.
- The brackets are designed with a built-in “prescription” to eliminate wire bends that had been necessary up to that time to compensate for differences in tooth anatomy.
- Variation in bracket thickness compensates for the varying thickness of individual teeth.
- Angulation of bracket slot relative to the long axis of the tooth allow for proper positioning of the roots.
- Torque in bracket slots compensate for inclination of facial surface of teeth.

Banding and Bonding

- 35–50% unbuffered phosphoric acid is used as an etching agent before direct bonding of orthodontic brackets.
- Tooth surface must not be contaminated with saliva, which promotes immediate remineralization, until bonding is completed.
- Glass ionomer cements are used to cement bands because of their fluoride releasing properties.



The four basic components of fixed appliances include bands, brackets, arch wires, and auxiliaries (elastics or ligatures to hold the archwire in brackets).

- Indications for using bands instead of brackets:
 - To better resist breakage, especially in areas of heavy mastication or cuspal interference.
 - For teeth that will need both a lingual and a labial attachment.
 - Teeth with short clinical crowns.
 - Tooth surfaces that are incompatible with successful bonding (e.g., amelogenesis imperfecta, stainless steel crown).

Archwire

- The properties of an ideal arch-wire:
 - High strength.
 - Low stiffness.
 - High range.
 - High formability.
- The stiffness of an orthodontic wire is a function of the length of the wire, the diameter, and the alloy composition.
- Various alloy composition of orthodontic arch-wires:
 - Stainless steel and cobalt chromium alloy.
 - Nickel titanium.
 - Beta-titanium (titanium and molybdenum alloy).

Possible Negative Sequella Associated with Orthodontic Treatment

- Orthodontic appliances may cause irritation or trauma to the gingiva or buccal or labial mucosa.
- They may act as plaque harbors.
- May interfere with proper oral hygiene.
- Prolonged orthodontic treatment is associated with root resorption.
- Prolonged orthodontic treatment is associated with decalcification stains on enamel surfaces.
- Orthodontic appliances increase the risk for gingivitis and caries.

► CROSSBITE AND OPEN BITE

Crossbite Definition and Etiology

- A crossbite is clinically observed as when teeth are on the wrong side of the opposing dentition.
- Crossbites can be skeletal, dental, or functional in origin.
 - A skeletal crossbite has smooth closure to centric occlusion.
 - A functional crossbite demonstrates a deviation in maxillary and mandibular midlines as the patient closes, often times a mandibular shift is noted.
- Crossbite may be associated with any of the following:
 - Heredity.
 - Maxillary/mandibular jaw size discrepancies such as reverse overjet scenarios sometimes observed in patients with class III skeletal tendencies where more than two maxillary anterior teeth are lingual to the mandibular anterior teeth.

- Oral habits such as a prolonged sucking habit.
- A labially situated supernumerary tooth, trauma, or arch length discrepancy.
 - Anterior crossbite of one or more of the permanent incisors is often associated with prolonged retention of a primary tooth.

Crossbite Classification

- Unilateral or bilateral.
- Anterior or posterior.

Crossbites can occur in any of the above the combinations, such as **bilateral posterior crossbite**.

- An anterior crossbite of multiple maxillary teeth in the primary dentition is often an indication of a skeletal growth problem or a developing class III malocclusion.
 - Children with class III tendencies will usually have end-on contact of the **primary** incisors, and will not develop the anterior crossbite until eruption of the **permanent** incisors.
- Posterior crossbites may be associated with a mandibular shift.

Crossbite Treatment

- Posterior crossbites and mild anterior crossbites need to be corrected in the first stage of treatment (as soon as possible), as the transverse dimension is the first to stop growth.
- Posterior crossbites are usually corrected with palatal expansion (see Fig. 5–7):
 - Rapid palatal expansion (RPE) devices are usually used.
 - Expander is activated two times a day (0.25 mm each turn); length of time will depend on amount of expansion desired.
 - After activation is completed, the expander remains in the mouth for 3–6 months for bone to form in the midpalatal suture region.

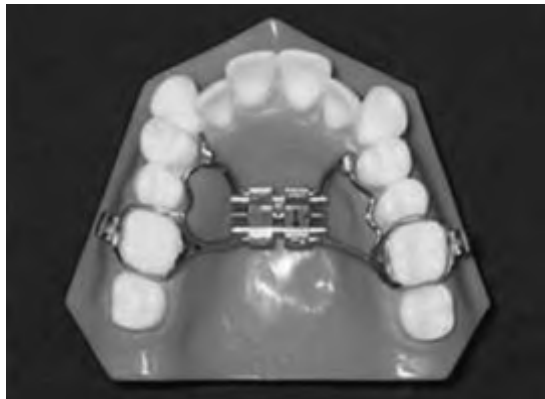


FIGURE 5–7. Rapid palatal expander.

(Courtesy of Dockstader Dental Lab, Fresno, CA, www.dockstader.com.)

- After palatal expansion:
 - Dentally: A diastema is observed between the central incisors. Diastema closure usually occurs spontaneously.
 - Skeletally: Expansion of nasal floor as a result of widening of the midpalatal suture.
- Mild cases such as anterior crossbite of one or more permanent incisors should be treated as soon as possible, otherwise complications such as loss of arch length can occur.
 - Be certain to create enough space mesiodistally for correction of anterior crossbite.
- Severe anterior crossbites are usually corrected in the second stage of conventional treatment.
- Corrected anterior crossbite is best retained by the normal incisor relationship.

Open Bite Definition and Etiology

- An open bite is a situation where the dentition in opposite arches cannot be brought into occlusion.
- Open bites can be skeletal or dental in origin.
- Anterior open bites are most commonly due to a finger habit.
 - Maxillary constriction results from increased pressure on the buccinator muscles from sucking.
 - As a result of the maxillary constriction from the finger habit, patients will have a tendency toward bilateral crossbite. They tend to shift the mandible to one side or another for better intercuspation, which may later on guide the eruption of posterior dentition (premolars and molars) into crossbite relationships.
 - Proclination of the maxillary incisors, retroclination of the mandibular incisors.
 - If the habit involves the hand resting on the chin, mandibular growth is sometimes retarded and produces a class II retrognathic profile.
- Compensatory tongue thrust habit is often observed in patients with open bites.

Open Bite Classification

- Open bite can be classified as:
 - Anterior open bite.
 - Posterior open bite.

Anterior open bites are more commonly seen in African Americans than Caucasians.

Open Bite Treatment

- Early management:
 - Habit control
- Orthodontic appliances (see Figs. 5–8A and B):
 - Tongue crib
 - Bluegrass



FIGURE 5-8A. Tongue crib.

(Courtesy of QC Orthodontics Laboratory Inc., Fuquay Varina, NC, www.qcortho.com.)



FIGURE 5-8B. Bluegrass appliance.

(Courtesy of QC Orthodontics Laboratory Inc., Fuquay Varina, NC, www.qcortho.com.)



FIGURE 5-9A. Transpalatal bar.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)

- Palatal expansion in cases of narrow maxillas.
- Palatal bars and lingual arches to reduce the vertical eruption of molars (see Figs. 5-9A and B).
- Posterior bite plates (see Fig. 5-10).
- High pull facebows (see Fig. 5-11).
- Myofunctional therapy in more severe cases.
- Orthodontic treatment only:
 - Usually limited in cases where the open bite is due to environmental factors.
 - More predictable treatment in patients with average or short lower facial height (average- to low-angle cases).



FIGURE 5-9B. Lower lingual holding arch.

(Courtesy of Dockstader Dental Lab, Fresno, CA, www.dockstader.com.)



FIGURE 5-10. Posterior bite plate.

(Courtesy of Precision Orthodontic Laboratory, North Hollywood, CA, <http://polusa.org>)

- Combined orthodontic and surgical treatment:
 - Required in most cases where skeletal open bites are involved, such as patients who show an excess of anterior vertical facial height, steep mandibular plane angle, or long lower facial heights.
- Retention:
 - Habit-control appliances such as tongue cribs can be included as part of the removal Hawley appliance.
 - Positioners can also be considered as part of retention because of their bite closing effect (see Fig. 5-12).



FIGURE 5-11. High-pull headgear.

(Courtesy of American Orthodontics, www.americanortho.com.)



Whenever possible, it is best to use fixed appliances as they do not require patient cooperation.



FIGURE 5-12. Positioners.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)

► SPACE MAINTENANCE

Types of Appliances

- Natural tooth is the best space maintainer.
 - Evaluate the patient's dental age and skeletal age if space maintenance will be needed in the case of a lost primary tooth.
 - If a broken down primary tooth is to be preserved, proper pulpal therapy in conjunction with a restoration would be needed.
- Space maintainer to replace one prematurely missing primary tooth.
 - Band and loop—prevents mesial migration of the primary second molar after unilateral loss of the primary first molar (see Fig. 5-13).



FIGURE 5-13. Band and loop space maintainer.

(Courtesy of JR Orthodontic Laboratory, San Diego, CA, www.jrortholab.com.)



FIGURE 5-14. Distal shoe.

(Courtesy of QC Orthodontics Laboratory Inc., Fuquay Varina, NC, www.qcortho.com.)

- Distal shoe—used when the primary second molar is lost prior to the eruption of the permanent first molar (see Fig. 5-14).
- Space maintainer to replace multiple missing primary teeth.
 - Lingual arc/arch—maintains space after multiple primary teeth are missing and the permanent incisors are erupted (see Fig. 5-9B).
 - Nance/transpalatal appliance (see Fig. 5-15):
 - Used for bilateral loss of primary maxillary molars.
 - Prevents mesial rotation and mesial drift of the permanent maxillary molars.
- Partial denture—for bilateral posterior space maintenance prior to eruption of eruption of permanent incisors.



FIGURE 5-15. Nance appliance.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)

Consequences of Early Loss of Primary Dentition

- Early loss of a primary maxillary second molar will result in a class II molar relationship on the affected side if space is not maintained.
- Premature loss of a primary canine may be due to arch length deficiency and result in lingual collapse of the mandibular anteriors.

Management of Early Loss of Teeth

- Early loss of primary second molar:
 - The most rapid loss of arch perimeter.
 - Due to **mesial tipping and rotation** of the **permanent first molar** after removal of the primary second molar.
 - When primary second molar is lost, **always maintain space** until arrival of the second premolar.
 - Space maintainer can be removed as soon as the permanent tooth begins to erupt through the gingiva.
 - The most reliable indicator of readiness of eruption of a succedaneous tooth is the extent of root development determined by radiographic evaluation.
 - $1/2-3/4$ root formation indicates succedaneous tooth mature enough for eruption.
- Loss of permanent first molar before eruption of permanent second molar:
 - Allow eruption and mesial drifting of the permanent second molar naturally.

► RETENTION

Fixed

- Mandibular lingual bonded retainer (see Fig. 5–16):
 - Often bonded canine to canine.
- Palatal bonded retainer:
 - Not used as frequently due to occlusal interference.
 - Good in cases where persistent spacing remains, such as midline diastemas.

Removable

- Conventional wire and acrylic :
 - Wrap-around (see Fig. 5–17A).
 - Standard Hawley (see Fig. 5–17B).
- Vacuum-formed (Essix) (see Fig. 5–18):
 - Rapid, economical, and esthetic.



FIGURE 5-16. Mandibular lingual bonded retainer.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)

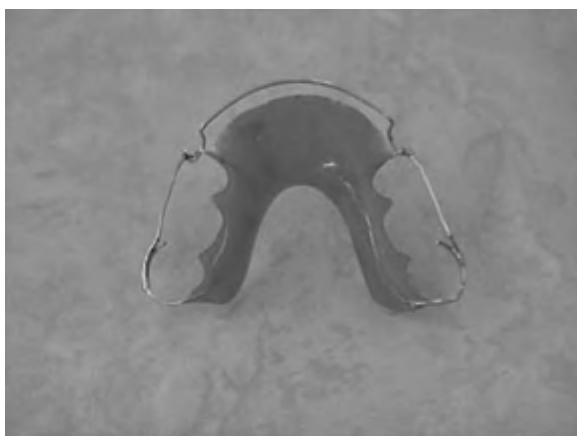


FIGURE 5-17A. Wrap-around Hawley.

(Courtesy of JR Orthodontic Laboratory, San Diego, CA, www.jrortholab.com.)



FIGURE 5-17B. Standard Hawley.

(Courtesy of JR Orthodontic Laboratory, San Diego, CA, www.jrortholab.com.)



FIGURE 5-18. Essix retainer.

(Courtesy of Dynaflex Laboratories, St. Louis, MO, www.dynaflex.com.)

Rationale for Retention

- Minimize changes due to growth.
- Maintain teeth in unstable conditions.
- Allow for reorganization of the gingival and periodontal tissue.
 - After malposed teeth have been moved into the desired position, they must be mechanically supported until the hard and soft tissues have been thoroughly modified both in structure and in function to meet the demands of the new position.
 - Occlusal result of **hard tissue** is modified by orthodontic treatment.
 - Occlusal result of **soft tissue** is modified and maintained by retention.
- Retention can be accomplished with either fixed or removable retainers.
- **Anterior crossbite** is easily retained after orthodontic correction by the overbite achieved during treatment.

Postorthodontic Circumferential Supracrestal Fibrotomy

- Indicated for **rotated maxillary lateral incisor** because the supraalveolar tissue is significantly responsible for the relapse of orthodontically rotated teeth.
- **Procedure:** Incision in sulcus is made to the crest of the bone where all to the collagen fibers are inserted into the root of the tooth.
 - Eliminate potential for relapse due to collagen fiber retraction.
 - Allow new fibers to form that will help retain the tooth in its new position.

Primary Molar Teeth Relationship

- This relationship determines the future antero-posterior position of the permanent first molars (see Fig. 5–19).
 - **Flush terminal plane** is the normal relationship. This causes edge-to-edge position of cusps of permanent maxillary and mandibular first molars. They will become class I by molars drifting forward (**early mesial shift**: mandibular molar twice as far as maxillary molar).
 - **Distal step** is equivalent of Angle's class II.
 - **Mesial step** is equivalent of Angle's class I.
- Angle class III is almost never seen in primary dentition due to the normal pattern of craniofacial growth in which the mandible lags behind the maxilla.



One of the most common causes of malocclusion is inadequate space management following early loss of primary teeth.

Primate Space

- Maxillary arch: Between lateral incisors and canines.
- Mandibular arch: Between canines and first molars.
- Normally present from the time teeth erupt.
- Generalized spacing of primary teeth is a requirement for proper alignment of the permanent incisors.
- Generalized spacing is most frequently caused by the growth of dental arches.

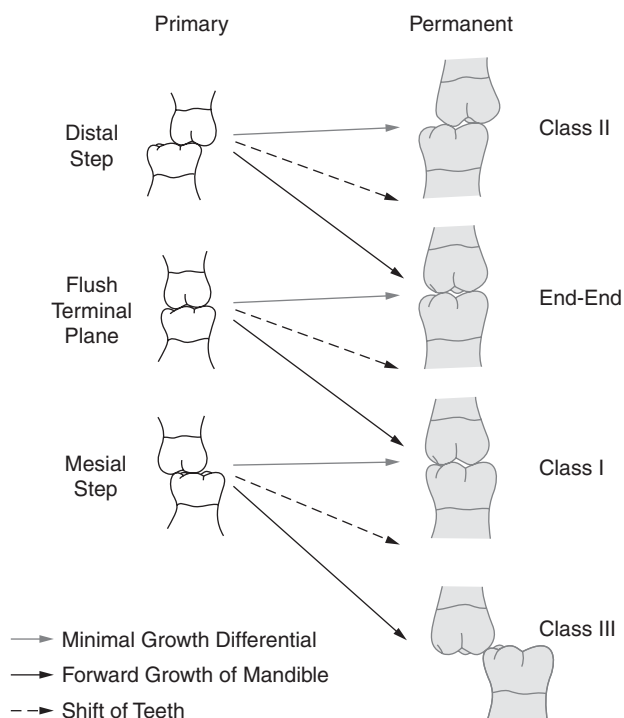


FIGURE 5–19. Primary molar teeth relationship.

- If spacing is present, adjacent teeth **may** drift if there is a loss of a primary incisor.
- If there is no spacing present and primary anterior teeth were in contact before the loss, there is usually a collapse in arch after the loss of one of the primary incisors.
- However, space closure occurs rapidly in the case of a lost permanent incisor. Thus space maintenance is indicated.

► MIXED DENTITION

Mixed Dentition Analysis

- Used to predict the amount of crowding after the permanent teeth erupt based on a correlation of tooth size.
- Performed during mixed dentition.
- Performed with Boley gauge, study models, and a prediction table.
- Procedure for **mandibular arch**:
 1. Anterior region:
 - A. Measure the mesial–distal diameter of the mandibular incisors (lower central and lateral incisors) and add them together.
 - B. Measure the space available for the mandibular incisor (arch circumference in the mandibular incisor region over the alveolar ridge).
 - $B \text{ minus } A = C$
 - Negative number = Crowding in incisor region.
 2. Posterior regions (per side):
 - D. Measure the space available for the canine and premolars on each side of the arch.
 - E. Calculate from the prediction table the size of the canine and premolar.
 - $E \text{ minus } D \text{ on each side} = F \text{ \& } G$
 - Negative number = Crowding in this particular side of the arch.
 3. Overall:
 - C = Crowding/spacing in incisor region.
 - F = Crowding/spacing in right canine and premolar region.
 - G = Crowding/spacing in left canine and premolar region.
 - Add these three numbers together:
 - Negative number = Overall crowding.
 - Positive number = Overall spacing.
- Procedure for **maxillary arch**:
 1. Anterior region:
 - A. Measure the mesial–distal diameter of the maxillary incisors (lower central and lateral incisors) and add them together.
 - B. Measure the space available for the maxillary incisor (arch circumference in the mandibular incisor region over the alveolar ridge).
 - $B \text{ minus } A = C$
 - Negative number = Crowding in incisor region.
 2. Posterior region:
 - C. Measure the space available for the canine and premolars on each side of the arch.

D. Use **mandibular** incisors to predict the size of the maxillary canines and premolars due to the wide variation of sizes of maxillary incisors. Thus, mandibular incisors are deemed to be more reliable for such prediction.

- E minus D on each side = F & G
- Negative number = Crowding in this particular side of the arch.

3. Overall:

- C = Crowding/spacing in incisor region.
- F = Crowding/spacing in right canine and premolar region.
- G = Crowding/spacing in left canine and premolar region.
- Add these three numbers together
 - Negative number = Overall crowding
 - Positive number = Overall spacing



*Supervision of a child's
development of occlusion is
most critical at age 7–10.*

Diastema

- 98% of 6-year-olds have diastema.
- 49% of 11-year-olds have diastema.
- Cause of diastema:
 - Tooth-size discrepancy.
 - Mesiodens.
 - Abnormal frenum attachment.
 - Normal stage of development.
- Treatment:
 - Spaces tend to close as permanent canines erupt:
 - However, the greater amount of spacing, the less likely the diastema space will close on its own.
 - Diastema usually closes spontaneously if space is 2 mm or less and the lateral incisors are in good position.
 - If diastema is caused by an abnormal frenum:
 - Align teeth orthodontically first.
 - Followed by a frenectomy after permanent canines have erupted.
- Methods of closing diastema:
 - Lingual arch with finger spring.
 - Hawley appliance with finger spring.
 - Cemented orthodontic bands with intertooth traction.

► GROWTH AND DEVELOPMENT

Effects of Environmental Influences during Growth and Development

- Patients with excessive overbite or anterior open bite usually have posterior teeth that are infra- or supraerupted respectively.
- Nonnutritive sucking habit leads to malocclusion only if it continues during the **mixed dentition stage**.
- Negative pressure created within the mouth during sucking is **not** considered a cause of constriction of the maxillary arch.
- “Adenoids,” which lead to mouth breathing, cannot be indicted with certainty as an etiologic agent of a long-face pattern of malocclusion because studies show that the majority of the long-face population has no nasal obstruction.

- Tongue thrust swallowing:
 - The tendency to place the tongue forward as patient swallows.
 - Appears to originate from the need to attain an oral seal, especially in cases of anterior open bite.
 - Thus, tongue thrust swallow should be considered the result of displaced incisors, not the cause.

Growth of the Mandible

- Major site of growth: **Condylar cartilage**.
- Occurs by:
 - Proliferation of cartilage at the condyles.
 - Selective apposition and resorption of bone at the membrane surfaces of the mandible itself (remodeling).
- Resorption occurs along the anterior surface of the ramus.
- Apposition of bone occurs along the posterior surface of the ramus.
- Growth at the mandibular condyle during puberty usually results in an **increase in posterior facial height**.
 - Major site of **vertical growth** in the mandible.
 - Provide space between the jaws into which the teeth erupt (especially molars).
- Main growth thrust: **Upward and backward** process to fill in the resultant space to maintain contact with the base of the skull.
 - Meanwhile soft tissue carries the mandible forward and downward.
- Mandible can and does undergo more growth in the late teens than the maxilla.
- Timeline of growth:
 - In infancy:
 - Ramus is located at the approximately where the primary first molar will erupt.
 - Progressive posterior remodeling creates space for the second primary molar and then for the sequential eruption of the permanent molar teeth.
 - If this process ceases before enough space is created for eruption of the third permanent molars, they will become impacted.
 - At age 6, the greatest increase in size of the mandible occurs **distal to the first molar**.
- Late mandibular growth theory:
 - The tendency for mandibular anterior crowding (**late incisor crowding**) during the late teens and early twenties period.
 - Mandibular incisors move distally relative to the body of the mandible late in mandibular growth.
 - Also occurs in individuals with congenitally missing third molars.
 - Most critical variable = extent of late mandibular growth.

Growth of the Maxilla

- Deposition of bone in the tuberosity region → the increase in height and elongation of maxillary arch in the posterior direction.

Formation of Bone

- Begins in the embryo where mesenchymal cells differentiate into either fibrous membrane or cartilage leading to two paths of bone development:
 - **Intramembranous ossification:**
 - Takes place in membranes of connective tissue.
 - Osteoprogenitor cells in the membrane differentiate into osteoblasts.
 - This collagen matrix formed undergoes ossification.
 - Formation of **maxilla** and **mandible**.
 - **Endochondral ossification:**
 - Takes place within a hyaline cartilage model.
 - Cartilage cells (**chondrocytes**) are replaced by bone cells (**osteocytes**) where calcium and phosphate are deposited into the organic matrix that has been laid down.
 - Formation of short and long bones, and ethmoid, sphenoid, and temporal bones.

Growth of Bone

- **Appositional growth:** Growth by the addition of new layers on those previously formed.
 - This is how bone grows once it is formed.
- **Do not confuse this with formation of bone** (via intramembranous and endochondral ossification).

Growth of Cartilage

- Occurs in two ways:
 - **Appositional growth:**
 - Recruitment of fresh cells, chondroblasts, perichondral stem cells, and the addition of new matrix to the surface.
 - Perichondrium consists of a fibrous outer layer and chondroblastic inner layer.
 - **Interstitial growth:**
 - Mitotic division of and deposition of more matrix around the existing chondrocytes already established in the cartilage.
 - Does **not** occur in **bone growth**.

Growth of Alveolar Process

- Grows in height and length to accommodate the developing dentition.
- **Exists only to support teeth:**
 - If tooth fails to erupt → alveolar process will never form.
 - If tooth is extracted → alveolar ridge completely atrophies over time.

Eruption Patterns

- Incisors:
 - In both maxillary and mandibular arches, permanent incisor tooth buds lie **lingual and apical** to primary incisors.

- Canines:
 - Relative to primary **mandibular** canine, permanent **mandibular** canine erupt more **facially** but most often **right in line**. Thus, if there is lack of room for permanent canine to erupt, it can be displaced either lingually or labially.
 - Permanent teeth move **occlusally and buccally** during eruption.
 - During active eruption, there is apposition of bone **on all surfaces** of alveolar crest and on the walls of the bony socket.
 - Maxillary arch is slightly longer (~128 mm) than the mandibular arch (~126 mm).

Failure or Delayed Tooth Eruption Patterns

- Caused by the failure of the eruption mechanism itself.
- Systemic causes:
 - Hereditary gingival fibromatosis.
 - Down's syndrome.
 - Rickets.
 - **Hyperthyroidism** can result in **premature** exfoliation of primary teeth.
- Localized causes:
 - Congenital absence.
 - Abnormal position of the crest.
 - Lack of space in the arch (crowding).
 - Supernumerary teeth.
 - Dilacerated roots.

Ectopic Eruption

- When a tooth erupts in the wrong place.
- Most likely to occur in the eruption of **maxillary first molars and mandibular incisors**.
- Frequency:
 - Much more common in the **maxilla**.
 - Often associated with developing skeletal **class II** pattern.
 - 2–6% of population.
 - 60% of cases are corrected spontaneously.
- Ectopic eruption of the permanent first molar:
 - When erupting too mesially and can potentially damage the roots of the primary second molars.
 - If untreated:
 - Will cause crowding in the arch.
 - Will be more susceptible to decay.
 - If decay is detected, extract primary second molar immediately and place space maintainer.
 - Treatment: Brass wire placed between primary second molar and permanent first molar to tip the permanent tooth distally.
 - Ectopic eruption of mandibular lateral incisors.
 - May lead to transposition of the lateral incisor and canine.

Wrist-Hand Radiograph (See Fig. 5–20)

- Used in predicting the time of the pubertal growth spurt because physical maturity and skeletal development correlates well with jaw growth.
- Can be used to judge physiologic (development) age by looking at the ossification and development of:
 - Carpal bones of the wrist
 - Metacarpals of the hands
 - Phalanges of the fingers
- After sexual maturity, much less growth is expected.

Lateral Cephalogram (See Fig. 5–21)

- Can be used to evaluate whether growth has stopped or is continuing.

Supernumerary Teeth

- Extra teeth that are usually small, peg-shaped, and do not resemble the teeth normal to the site.
- Most common site: between central incisors. (It is called **mesiodens**.)
- 2:1 predilection for males.



FIGURE 5–20. Wrist-hand radiograph.

(Courtesy of Wikipedia, http://en.wikipedia.org/wiki/Image:X-ray_boy_hand.jpg.)



FIGURE 5-21. Lateral cephalometric radiograph.

(Courtesy of Dr. Abed Alkhatib, Division of Dentistry, Oral and Maxillofacial Surgery, New York Presbyterian Hospital, Cornell Campus.)

- They can cause:
 - Diastema or spacing between maxillary central incisors if they are mesiodens.
 - Crowding of normal teeth.
 - May delay the eruption of permanent teeth.
 - For example, inverted mesiodens can cause delayed eruption of maxillary central incisors.
- Treatment: surgically remove and observe progress of permanent teeth.
- Conditions associated with multiple supernumerary teeth:
 - Gardener's syndrome
 - Down's syndrome
 - Cleidocranial dysplasia
 - Sturge-Weber syndrome

Oligodontia

- Absence of one or more teeth
- More common in female than male
- Often associated with smaller than average tooth-to-size ratio

Serial Extraction (See Fig. 5-22)

- The orderly removal of selected **primary** and **permanent** teeth in a predetermined sequence.
- Indications: Severe class I malocclusion in mixed dentition that has insufficient arch length.
- Sequence: Primary canine → primary first molars → permanent first premolars.

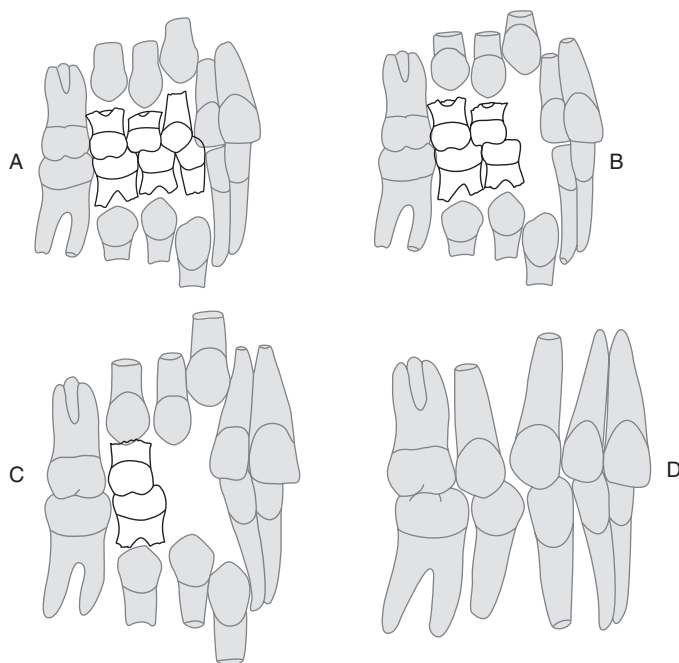


FIGURE 5-22. Serial extractions.

- **KEY:**
 - Extract first premolars before permanent canines erupt.
 - Should always use with a lingual arch in mandible and Hawley appliance in the maxilla for support and retention.
 - Usually followed by full orthodontic treatment
 - Severe arch space deficiency in permanent dentition (over 10 mm) will almost always require extractions to properly align teeth.

► ORTHODONTIC TERMS

Overbite

- The vertical overlapping of the maxillary anterior teeth over the mandibular anterior teeth.

Overjet

- The horizontal projection of the maxillary anterior teeth beyond the mandibular anterior teeth.

Physiologic Occlusion

- An occlusion that is not necessarily an ideal class I occlusion but one that adapts to stress of function and can be maintained indefinitely.



Permanent molars have a natural tendency to drift mesially.

Pathological Occlusion

- An occlusion that cannot function without contributing to its own destruction.
- May manifest itself by any combination of:
 - Excessive wear of the teeth without sufficient compensatory mechanisms.
 - TMJ problems.
 - Pulpal changes ranging from pulpitis to necrosis.
 - Periodontal changes.

Leeway Space

- Definition: The difference between:
 - Mesiodistal widths between **primary** canine + first molar + second molar
 - Mesiodistal widths between **permanent** canine + first premolar + second premolar
- **Mandibular** leeway space is approximately 3–4 mm.
- **Maxillary** leeway space is approximately 2–2.5 mm.
- During canine–premolar transition period, the permanent first molars generally move mesially into the leeway space after the primary second molars are shed → loss in arch length (a.k.a. **late mesial shift of a permanent first molar**).
- Permanent successors of primary first and second molars are most often smaller than their primary predecessors.

Orthodontic Movements

- **Extrusion:** The displacement of a tooth from the socket in the **direction of eruption**.
- **Intrusion:** Movement into the socket along the long axis of the tooth.
 - Difficult to accomplish.
- **Tipping:** The crown moves in one direction, while the tooth tip is displaced in the opposite direction due to rotation or pivoting of tooth around the **axis of rotation** (at the junction of the apical and middle 1/3 of root).
 - Accomplished most easily with anterior incisor teeth.
- **Translation (bodily movement):** Coupled force is applied to the crown to control root movement in the same direction as crown movement by applying force through the tooth's **center of resistance**.
 - Difficult to accomplish.
- **Torque:** Controlled root movement **labiolingually** or **mesiodistally** while the crown is held relatively stable.
 - Controlled root movement mesiodistally is also referred as **uprighting**.
- **Rotation:** Revolving the tooth around its long axis.
 - Recurrence occurs after orthodontic correction because of the persistence of the elastic **supracrestal gingival fibers** (free gingival and transseptal fibers mainly).
 - Collagen fibers are the primary components of attached gingiva.
 - Need long-term retention to prevent relapse.
- **Osteoclasts** are present on the side toward which the tooth is being moved.
- **Osteoblasts** are present on the side of the root from which the tooth moves.

Pediatric Dentistry

Paul Li, DDS

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Primary Dentition

FIRST DENTAL VISIT

- Evaluation period: ≤ 6 months of first tooth eruption and no later than first birthday.
- Dentist should perform dental and medical histories as well as oral exam.

NATAL TEETH

- Present at time of birth.
- 1 in every 2000–3000 births.
- Mandibular incisor region.
- Little root formation → attached to soft tissue makes them unstable.
- May cause trauma during nursing due to location.
- Treatment: Frequently removed due to fear of aspiration.

NEONATAL TEETH

- Present within the first 30 days after birth.
- Same incidence as natal teeth.
- Mandibular incisor region.
- Both natal and neonatal teeth have hypocalcified enamel matrix.

ERUPTION SEQUENCE OF PRIMARY DENTITION

- There are no bicuspid in primary dentition!
- Rule of 4:4 teeth every 4 months starting with four teeth at 7 months.

Age (Months)	Teeth Erupted	Specific Teeth
7	4	Mandibular incisors
11	8	Maxillary incisors
15	12	First molars
19	16	Canines
23	20	Second molars

PRIMARY ERUPTION AND EXFOLIATION

Maxillary (Mx)

	Eruption (Months)	Exfoliation (Years)
Central incisor (CI)	8–12	6–7
Later incisor (LI)	9–13	7–8
Canine (K9)	16–12	10–12
First molar	13–19	9–11
Second molar	25–33	10–12

Mandibular (Mn)

	Eruption (Months)	Exfoliation (Years)
Central incisor (CI)	6–10	6–7
Later incisor (LI)	10–16	7–8
Canine (K9)	17–23	9–12
First molar	14–18	9–11
Second molar	23–31	10–12

GENERAL PRIMARY TEETH CHARACTERISTICS

- Uniform enamel thickness of 1 mm occlusally
- Short crowns
- Exaggerated buccal and lingual cervical ridges
- Constricted cervical area
- Narrow faciolingually from an occlusal view
- Shallow anatomy
- Prominent mesial cervical ridge
- Longer/slender and divergent roots with little or no trunk

CHARACTERISTICS OF PRIMARY MANDIBULAR FIRST MOLAR

- Resemble no other teeth
- Largest/longest = mesial
- Wide mesial distally with a pronounce cervical ridge (CR)
- Mesial marginal ridge (MMR) $\approx$ cusp
- Big MB cervical ridge
- No central fossa
- Difficult class II preparation

Permanent Dentition

PERMANENT ERUPTION GUIDELINE

- Mn K9 = Mn 9–10; thus, Mx = 11–12 (Mn before Mx)
- Correlate the time of permanent teeth eruption to primary teeth exfoliation

ERUPTION SEQUENCE OF PERMANENT DENTITION

- Mx: 1st molar $\rightarrow$ CI $\rightarrow$ LI $\rightarrow$ 1st PM $\rightarrow$ 2nd PM $\rightarrow$ K9 $\rightarrow$ 2nd M $\rightarrow$ 3rd M
- Mn: 1st molar $\rightarrow$ CI $\rightarrow$ LI $\rightarrow$ K9 $\rightarrow$ 1st PM $\rightarrow$ 2nd PM $\rightarrow$ 2nd M $\rightarrow$ 3rd M

Range in Years	Teeth
6–7	All first molars <i>Mn</i> Central incisor
7–8	<i>Mx</i> CI <i>Mn</i> LI
8–9	<i>Mx</i> LI
9–10	<i>Mn</i> canine
10–12	All premolars (PM)
11–12	<i>Mx</i> canine
11–13	All second molars
17–21	All third molars

Angle Classification of Malocclusion

Class	Molar Relationship	Canine Relationship	Description
I/Neutroocclusion (Fig. 6-1)	Mx first molar MB cusp → Mn first molar buccal groove	Mx canine → Distal Mn canine and mesial Mn first PM	■ “Normal” ■ Mesognathic (Fig. 6-4)
II/Distocclusion (Fig. 6-2)	Mn first molar buccal groove is DISTAL to Mx first molar MB cusp	Distal surface of Mn canine is distal to the mesial of Mx canine	■ Retrognathic ■ Div I: Mx teeth in labioversion/overjet ■ Div II: Linguoversion of Mn teeth
III/Mesioocclusion (Fig. 6-3)	Mn first molar buccal groove is MESIAL to Mx first Molar MB cusp	Distal surface of Mn canine is mesial to the mesial of Mx canine	■ Prognathic

Malocclusions

Term	Definition
Anterior crossbite (Fig. 6-5)	Maxillary incisors are positioned lingually against mandibular incisors in occlusion.
Posterior crossbite (Fig. 6-6)	Primary or permanent maxillary teeth that are occluded lingually to the opposing mandibular teeth. Can be unilateral and or bilateral.
Edge-to-edge bite (Fig. 6-7)	Incisal edges of maxillary and mandibular teeth meet.
End-to-end bite	Cusp-to-cusp occlusion of posterior teeth
Open bite (Fig. 6-8)	Failure of maxillary and mandibular teeth to occlude.

**FIGURE 6-1. Neutroocclusion.**(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)



FIGURE 6-2. Distoclusion.

(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)



FIGURE 6-3. Mesioclusion.

(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)



FIGURE 6-4. Mesognathic.

(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)



FIGURE 6-5. Anterior crossbite.

(Reproduced, with permission, from McGann BD, Orthodontic Distraction Osteogenesis: Part 2 Maxillary Labial Corticotomy to Improve Treatment of Skeletal Class III Cases, Surgically Assisted Orthodontics.)

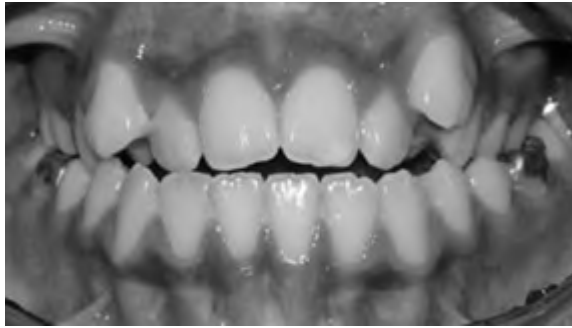


FIGURE 6-6. Posterior crossbite.

(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)



FIGURE 6-7. Edge-to-edge bite.

(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)



FIGURE 6-8. Open bite.

(Courtesy of Mohammad Yasser Tabbaa, DDS, MS, www.arabicsmile.com.)

Background

- Use for prevention and control of caries.
- Most cost-effective way is through drinking water supply.
- After 6 months of age, fluoride can be effective in primary preventive procedures.

Home fluoride programs using fluoride mouth rinses or brush-on fluoride gels should be recommended for use by school-aged child at high risk for caries. If a patient at high risk for caries cannot or will not comply with home fluoride therapy, frequent professional fluoride treatments may be substituted.

High Risk for Caries

- Children with orthodontic and or prosthodontic appliances
- Children with reduced salivary function
- Children who are unable to clean their teeth properly
- Children with dietary risk
- Children with mothers and siblings with history of extensive caries
- Children with family history of having high oral levels of cariogenic bacteria
- Children with active caries

SUPPLEMENTAL FLOURIDE RECOMMENDATION

Age	<0.3 ppm	0.3–0.6 ppm	>0.6 ppm
Birth–6 months	0	0	0
6 months–3 years	0.25 mg	0	0
3–6 years	0.5 mg	0.25 mg	0
6 years upto at least 16 years	1.0 mg	0.5 mg	0

Fluoride Facts

- Food and Nutrition Board recommends public water supplies be fluoridated when levels are significantly below 0.7 mg/L.
- Fluorine intake of 20–40 mg/day can inhibit the important enzyme phosphatase.
- Phosphatase is needed for calcium utilization/metabolism in tissues including the bones and teeth.
- Fluorine intake of 40–70 mg/day can cause heartburn and pains in the extremities.

Symptoms of Fluoride Toxicity

- Nausea
- Vomiting
- Hypersalivation
- Abdominal pain
- Diarrhea
- Acute → cardiac failure and respiratory paralysis
- Fluorosis (“mottled enamel”)

Treatment of Fluoride Toxicity

- Syrup of ipecac to induce vomiting
- Calcium-binding products (i.e., milk/milk of magnesia) will lower the acidity of the stomach → insoluble complexes with fluoride and ↓ absorption
- Call 911 if necessary

► PEDIATRIC PATHOLOGY

Odontogenic Development

- Lobes = primary center of ossification of the tooth.
- Lobes are separated by developmental grooves in posterior teeth.
- Lobes are separated by developmental depressions in anterior teeth.
- All anterior teeth have four lobes (three labial and one cingulum).
- Premolars have three buccal lobes and one lingual lobe (Mn second PM = 3 buccal + 2 lingual).
- First molars have five lobes (one for each cusp).
- Second molars have four lobes (one for each cusp).
- Third molars have at least four lobes.

Tooth Histogenesis (Chronologic Order)

- Ectomesenchyme influences the downgrowth of oral epithelium → tooth germ.
- Elongation of inner enamel epithelial cells of enamel organ.
- Differentiation of odontoblasts.
- Deposition of the first layer of dentin.
- Deposition of the first layer of enamel.
- Deposition of root dentin and cementum.

Life Cycle of a Tooth (Chronologic Order)

- Initiation (bud stage): Interaction of oral epithelium ectomesenchyme → dental lamina.
- Proliferation (cap stage): Formation of the shape of the tooth and enamel organ.
- Differentiation (bell stage): Differentiation into specific tooth tissue types, histodifferentiation.
- Apposition: Cells from the bell stage begin depositing their corresponding tissues.
- Calcification: Primary teeth begin calcification during second trimester.
- Eruption: Emergence of tooth through gingiva.

Fusion vs. Gemination vs. Concrescence

Fusion	Gemination	Concrescence
■ Two tooth buds joined together. ■ Evident of dentin contact! ■ Appear as a macrodont (large crown). ■ Usually with two root canals. ■ Usually seen in incisor areas. ■ If the tooth count is reduced, it is fusion.	■ One tooth bud splits. ■ See incomplete formation of two teeth. ■ Two crowns on a single root. ■ Hard to differentiate vs. fusion. ■ Usually in incisor region. ■ If the tooth count is normal, it represents germination.	■ A form of fusion. ■ Cementum contact! ■ Occur after root formation completed. ■ Arise as a result of trauma.

Attrition vs. Abrasion vs. Erosion

Attrition (Fig. 6–9)	Abrasion (Fig. 6–10)	Erosion (Fig. 6–11)
■ Physiologic wearing away of tooth structure. ■ Normal aging consequence. ■ Seen on incisal edges, occlusal surfaces, and proximal surfaces of teeth. ■ Often present as flatten incisal edges. ■ Example = bruxism.	■ Pathologic wearing away of tooth structure due to mechanical forces. ■ Toothbrush-abraded tooth presents with a clinical V-shaped ditch on the affected surface. ■ Also result from abnormal mechanical habits.	■ Chemical loss of tooth structure. ■ Labial surfaces of patients who ingest large amounts of highly acidic beverages, or, in people who suck on citrus fruits such as lemons, oranges, etc. ■ Lingual surfaces of patients with chronic vomiting habits, particularly those suffering from anorexia nervosa. ■ Eroded tooth usually displays a shallow smooth scooped-out depression.



FIGURE 6–9. Attrition.

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)



FIGURE 6-10. Abrasion. (Also see color insert)

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)



FIGURE 6-11. Erosion.

(Courtesy of Marquette University School of Dentistry.)

Enamel Hypocalcification vs. Enamel Hypoplasia

Enamel hypocalcification	Enamel hypoplasia (Fig. 6-12)
■ Normal quantity but bad quality ■ Defective maturation of ameloblast ■ Defect in mineralization matrix ■ Chalky teeth and yellow to brown ■ Affects both deciduous and succedaneous teeth	■ Normal quality but deficient quantity of enamel ■ Defective enamel matrix formation ■ No contact between teeth ■ Affects both deciduous and succedaneous teeth ■ Hereditary and environmental form ■ Seen in children with hypoparathyroidism

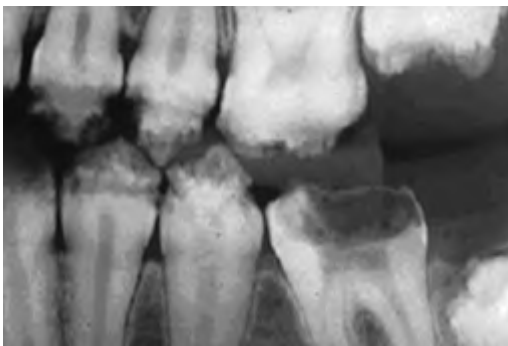


FIGURE 6-12. Enamel hypoplasia.

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)

Amelogenesis Imperfecta

- Hereditary form of enamel hypoplasia.
- Involves the complete primary and permanent dentitions.
- Inherited dominant trait → soft and thin enamel.
- Teeth appear yellow to brown due to the thin enamel and dentin showing through (Fig. 6-13).
- With a healthy enamel covering teeth are easily damage and susceptible to decay.
- Dentinal tissue and pulpal tissue are not affected.
- Teeth can be treated with full crowns since only the enamel is affected.
- Don't get confused with dentinogenesis imperfecta.
- Radiographic you will see teeth similar to crown preparation (Fig. 6-14).

Dentinogenesis Imperfecta

- Inherited dominant trait
- Undermineralization of dentin
- Affect both deciduous and permanent teeth
- Gray to brown appearance: “opalescent hue” (Fig. 6-15)
- No pulp cavity! (Fig. 6-16)
- Bulbous crowns with short roots
- Type I associated with osteogenesis imperfecta (blue sclera, fragile bones, hearing loss)
- Type II most common
- Type III have multiple pulpal exposures in primary dentition



FIGURE 6-13. Amelogenesis imperfecta clinical photograph. (Also see color insert)

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)

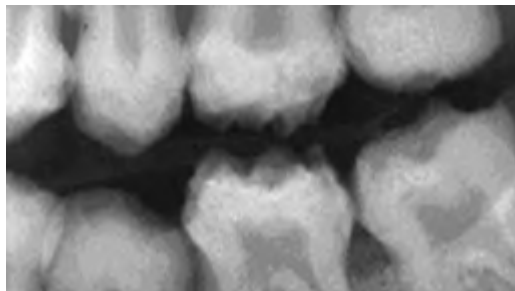


FIGURE 6-14. Amelogenesis imperfecta film.

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)

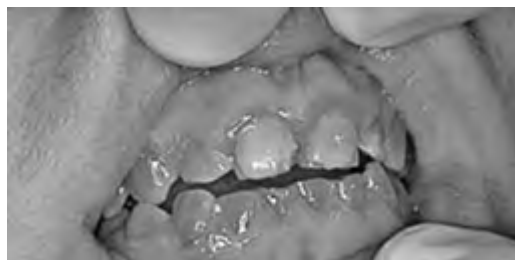


FIGURE 6-15. Dentinogenesis imperfecta clinical photograph. (Also see color insert)

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)



FIGURE 6-16. Dentinogenesis imperfecta film.

(Courtesy of Columbia University College of Dental Medicine, Teaching Slides.)

Early Childhood Caries (ECC)

- Formerly known as nursing bottle syndrome.
- Defined as rampant decay that results from sleeping with feeding bottle.
- Most common deciduous teeth = maxillary incisor.
- General involvement = maxillary anterior teeth.
- Also known as = baby bottle tooth decay (BBTD), bottle-mouth syndrome, early childhood caries, nursing caries, bottle caries, and infant caries.
- Encourage infants to drink from a cup prior to first birthday.
- Supplement infant's diets with nonliquid 4–6 months of age.
- Serve liquids with sugar from a cup.
- Initiate oral hygiene with the eruption of the first primary tooth.
- First dental visit within the first 6 months of eruption of first tooth.

Acute Necrotizing Ulcerative Gingivitis (ANUG)

- Usually in people of 15–35 years old
- Also known as Vincent's infection, Vincent's angina, and trench mouth
- Symptoms = painful hyperemic gingival, punched out erosions covered by gray pseudomembrane, and fetid odor
- Risks = bad oral hygiene, poor nutrition, smoking, and stress
- Bacteria = Fusiform, Spirochetes, and *Prevotella intermedia*
- Rare in preschool children
- Treatment include: debridement, hydrogen peroxide rinses, and antibiotics

► SYSTEMIC PATHOLOGY

Achondroplasia

- Form of short-limb dwarfism
- Equal frequency in male and females
- Large head with short arms and legs compared to trunk length
- Deficient cranial base growth
- Small maxilla
- Commonly will class III malocclusion
- Delayed eruption and exfoliation of teeth
- Underdeveloped mandible

Gigantism

- Condition characterized by excessive height growth
- Rare condition of pituitary gigantism due to prepubertal growth hormone excess
- Oversecretion by a group of somatotrope cells of the anterior pituitary gland
- Enlarged tongue
- Mandibular prognathism
- Longer roots

Gingivostomatitis

- Herpes simplex virus-1 (HSV-1) infection → sores of mouth and gingival.
- Acute/primary form generally involves children under age of 3.
- Prodromal symptoms (fever, malaise, irritability, headache, dysphagia, vomiting, lymphadenopathy) of 1–2 days before appearance of ulcers.
- Treatment: relief of symptoms and maintain nutritional and fluid intake.
- Sequelae: Recurrent herpes labialis, HSV-1 antibodies.

Coxsackie Virus

- Painful white/yellow ulcers with bright red surroundings.
- Cause aphthous ulcers.
- Trigger by stress, menstrual cycles, hormonal changes, allergies.
- Begin with tingling or burning sensation.
- Pain ↓ in 7–10 days with complete healing 1–3 weeks.
- Recurrent ulcers are primary on mucosa while herpetic lesions on tissue-bound periosteum.
- Recurrent aphthous minor: less than 1 cm in diameter, last ≈2 weeks.
- Recurrent aphthous major: over 1 cm in diameter, last ≥2 weeks, and heal with scarring.
- Recurrent herpetiform: cluster of ulcers.
- Frequent recurrences of ulcers should be screened for diabetes mellitus or Behcet's syndrome.
- Treatment include: topical steroids (Triamcinolone).

Cretinism

- Severe hypothyroidism
- Characterize by defective mental and physical development
- Dwarfed bodies with curved spines and pendulous abdomen
- Limbs are distorted
- Harsh and scanty hair
- Under developed mandible
- Over developed maxilla
- Enlarged tongue
- Delayed eruption of teeth
- Retained primary teeth
- Adult hypothyroidism = myxedema

► CRANIOFACIAL ANOMALIES

Facial Clefts

- Cleft lip (CL) and cleft palate (CP) account for half of the total number of defects.
- CL = separation of the two sides of the lip.
- CP = an opening in the roof of the mouth which the two sides of the palate did not join.
- Both CL and CP can be unilateral or bilateral.
- Unilateral CL and CP is an isolated nonsyndromic birth defect.

- CL- and CP-associated syndromes include Stickler's, Vander Woude's, and DiGeorge syndrome.
- Chances of a family having more than one child with a cleft after having a child with unilateral CL and CP is approximately 2–4%. (Keep in mind incidences do increase with additional family history and bilateral clefts.)

Cleft Palate (CP)

- Occur in approximately 1 in 700 live births (CL and CP).
- CP alone is seen in approximately 1 in 2000 live births.
- Occurs during sixth to eighth week in utero.
- Isolated clefts are more common in females.
- Fissure in the midline of the palate due to failure of fusion of the two sides of the palate.
- Impaired speech and swallowing.
- Inability for soft palate to close the air flow into the nasopharynx.

Cleft Lip (CL)

- Occurs during fifth to sixth week in utero
- Failure of maxilla and frontonasal processes to merge
- More common in males
- More frequent on the left side than the right

Four Classes of CL

- Class I: Unilateral notching of vermillion not extending to lip
- Class II: Same as class I but extending into lip but not to floor of the nose
- Class III: Class II + extending into floor of the nose
- Class IV: Any bilateral cleft of the lip

Timing of Treatment for CL and CP

- CL repair should occur 10 weeks after birth.
- CP repair should occur 9–18 months after birth.
- Pharyngeal flap or pharyngoplasty 3–5 years or later depending on speech development.
- Alveolar reconstruction 6–9 years based on dental development.
- Cleft orthognathic surgery 14–16 years in girls and 16–18 years in boys.
- Cleft rhinoplasty surgery depends on skeletal maturity.
- CL revision at anytime but best after age 5.

Rule of 10's for timing of CL repair

Infant should be:
 ≥10 weeks old,
 ≥10 g/dl hemoglobin, and
 ≥10 lbs.

Pierre Robin Syndrome

- Collagen gene 2A1 mutation
- Hypermobility of joints

- Mitral valve prolapse
- Micrognathia leading to breathing and feeding difficulties
- Glossoptosis
- High arched CP

Treacher Collins Syndrome

- Also called mandibulofacial dysostosis
- Mutation of 5q32 gene
- Downslanting of eyes
- Notched lower eyelids
- Midface developmental deficiency
- Underdeveloped, malformed, and/or prominent ears

Down Syndrome

- Low caries susceptibility.
- Trisomy 21.
- Delayed physical and mental development.
- Delayed eruption of teeth.
- Short and stocky stature.
- Face is broad and flat, with slanting eyes, and a short nose.
- Ears are small and low set.
- Heart defects are common! (Check for prophylactic requirement.)

Confusing “OMAS”

Hemangioma	Lymphangioma	Neurofibroma
■ Most common benign tumor of infants ■ Due to proliferation of blood vessels ■ Growth is independent of the growth of the child ■ Most appear within 1–2 weeks after birth ■ 5 times more in girls ■ Frequent on lips, tongue, and buccal mucosa ■ Often self-involute or can be surgically removed	■ Well-circumscribed mass of lymphatic vessels ■ Most in neck and axilla ■ Are red to blue translucent masses that seem spongy ■ Treated by excisional biopsy	■ Moderately firm and encapsulated ■ Proliferation of Schwann cells ■ On tongue, buccal mucosa, vestibule, and palate ■ 5–15% may become malignant ■ Multiple lesions need to consider neurofibromatosis (Von Reckling-Hausen’s)

Confusing “BUMPS”

Measles	German Measles	Small Pox	Mumps
Called Rubeola. Paramyxovirus. 1–2-weeks incubation. Sx: fever, cough, a rash. Koplik’s spots intraorally. Highly contagious viral infection. Supportive treatment.	Called Rubella. Benign viral disease. Sx: red bump, swollen lymph nodes, mild fever, and enamel defects. Supportive treatment.	Acute viral disease. Sx: high fever, nausea, vomiting, chills, and headache. Ulcerated/vesicular lesions. Variola major is the severe and most common form of smallpox, more extensive rash, and higher fever. Variola minor is a less common presentation of smallpox, and a much less severe disease. Best to vaccinate.	Acute and contagious. Unilateral/bilateral swelling of salivary gland (parotid) Replicates in the upper respiratory tract. Spread by contact with respiratory secretions, saliva, and fomites. Incubation time = 12–25 days. Vaccinate with MMR (measles, mumps rubella).

► CLINICAL MANAGEMENT

Behavior Management Techniques

- Voice control.
- Positive reinforcement versus negative reinforcement.
- Distraction.
- Tell-show-do (most important).
- Hand-over-mouth (consent is necessary).
- Nonverbal communication.
- Sedation.
- Mentally challenged children can be treated like their peers but keep appointments brief.
- Keep in mind we are all fearful of the unknown!

► TRAUMA MANAGEMENT

Types of Tooth Trauma

- **Concussion:**
 - No injury of supporting structures.
 - No evidence of displacement of tooth/teeth.
 - Clinically tooth will be tender to exam.
 - No sign of mobility upon examination.

Primary Teeth	Permanent Teeth
■ No pulpal therapy unless evidence of infection ■ Minimal risk of pulpal necrosis	■ High chance of pulpal necrosis with closed apices

- **Subluxation:**
 - Injury of supporting structures
 - Have loosening of tooth
 - No displacement of tooth
 - Clinically = mobile tooth without displacement
 - May have sulcular hemorrhage

Primary Teeth	Permanent Teeth
■ Observe for possible pathology ■ Should be normal within 2 weeks	■ Stabilization ■ Occlusal enameloplasty as needed ■ Splint for no more than 2 weeks ■ High percent of pulpal necrosis

- **Lateral Luxation:**
 - Displacement of tooth beside in an axial direction.
 - Torn periodontal ligaments with contusion or fracture of alveolar bone.
 - Clinically, you have a laterally displaced tooth.
 - Usually nontender and nonmobile.
 - Increase in periodontal space.

Primary Teeth	Permanent Teeth
■ Allow passive repositioning ■ Active repositioning with splinting for 1–2 weeks ■ Tooth requiring reposition will more likely have pulpal necrosis versus spontaneous reposition	■ Reposition as soon as possible and stabilize ■ Splint for 2–4 weeks ■ High risk of pulpal necrosis ■ High risk of root resorption

- **Intrusion:**
 - Apical displacement into alveolar bone.
 - Compression of periodontal ligament is evident.
 - Usually have crushing fracture of alveolar socket.
 - Clinically, tooth appears short or cannot be found intraorally.

Primary Teeth	Permanent Teeth
■ Allow spontaneous reeruption ■ Extraction when it is endangering the permanent tooth/tooth bud ■ Most will reerupt in 2–6 months ■ Risk of ankylosis	■ Reposition to anatomically correct location ■ Stabilize and initiate endodontic therapy within 3 weeks of incident ■ High risk of pulpal necrosis and root resorption

- **Extrusion:**
 - Partial displacement out of the socket axially.
 - Torn periodontal ligaments.
 - Clinically tooth appears elongated and mobile.

Primary Teeth	Permanent Teeth
■ Reposition unless indicated for extraction ■ Splint for 1–2 weeks	■ Reposition as soon as possible ■ Splint for 3 weeks ■ High risk of pulpal necrosis

- **Avulsion:**
 - Complete displacement out of socket.
 - Severe periodontal ligaments.
 - Clinically missing tooth.

Primary Teeth	Permanent Teeth
■ Should NOT be replanted	■ Replant as soon as possible and stabilize for 1–2 weeks ■ Consider antibiotic and tetanus coverage ■ Storing solution in order of preference: 1. Viaspan 2. Hank's solution 3. Cold milk 4. Saliva (buccal vestibule) 5. Saline 6. Water

Ellis Classification

Class I	Class II	Class III	Class IV
Involves little or no dentin. Initiate enameloplasty and or bonding.	Involves dentin but no pulp. Cover exposure with calcium hydroxide or glass ionomer type restoration.	Pulp exposure. Initiate appropriate pulp therapy and restoration. A primary tooth will often lead to pulpal necrosis.	Lost of entire crown. Initiate pulpectomy followed by stainless steel crown.

Note: Root fractures of primary teeth are treated with the same guidelines as adult teeth. Keep in mind that the pulpal prognosis is less favorable.

- **Pediatric mandible fracture:**
 - Most should be treated conservatively.
 - Often present as greenstick or incomplete fracture.
 - Mandible fractures account for 20–50% of all childhood fractures.
 - Fractures are more common in boys.
 - Large number of patients also concomitantly present with intra-abdominal, neurocranial, and or orthopedic injuries.
 - Condylar fracture incidence decreases with increase in age.
 - Body and angle fractures increase with increase in age.

Treatment of Pediatric Mandible Fracture

Surgical	Nonsurgical
■ Interdental wiring (2–3 weeks). ■ Occlusal splints with circummandibular wires (2–3 weeks) ■ Drop wires and circummandibular wires ■ Sutures with or without splints ■ Open reduction and internal fixation	Clinical evidence suggests minimal displaced greenstick fracture in children with little or no intervention. Conservative management prevent muscular atrophy, ankylosis, and allow better oral hygiene.

► PHARMACOLOGIC MANAGEMENT

- **Oral Sedation with chloral hydrate:**
 - Acts on CNS to induce sleep.
 - Will not affect breathing, blood pressure, and reflexes.
 - Onset of 15–30 minutes given by mouth.
 - Children will often be irritable and excited prior to becoming sedated.
 - Classified as sedative-hypnotics.
 - 25–100 mg/kg orally (maximum single dose = 1 g and not exceed 100 mg/kg or 2 g).
 - Contraindicated in marked hepatic or renal impaired patients.
 - Active metabolite = Trichloroethanol.

Pediatric Local Anesthesia

pKa	Lipid Solubility	Protein binding
■ Affects onset ■ Lower values = rapid onset of action	■ Affects anesthetic potency ■ Higher values = increased potency	■ Affects duration ■ Higher values = increased duration of action

- Maximum pediatric dose = (weight of child in pounds/150) × (maximum adult dose in milligrams).
- Safest local anesthetic to use in pediatrics is 2% lidocaine with 1:100,000 epinephrine.
- Simple method for remembering pediatric dose with or without vasoconstrictor = one cartridge for every 20 lb.

Endodontics

Brian Jeon, DMD

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Medical and Dental History

- **History of pain:** Describe intensity, type, timing, location, duration, and modifying factors of pain. That is, the patient complains of severe, throbbing, intermittent pain in lower right quadrant for 3 days, and the pain is exacerbated by cold drinks and chewing.
- Include history of swelling, sinus tract, recent dental care, and dental trauma.
- Identify the source of patient's chief complaint.

Clinical Exam

- **Extraoral exam:**
 - Check general appearance, skin tone, and facial asymmetry
 - Note any swelling, redness, sinus tracts, tender or enlarged lymph nodes, or tenderness upon palpation
- **Intraoral exam:**
 - Examine the mucosa and gingiva visually and digitally for discoloration, inflammation, ulceration, swelling, and sinus tract
 - Provide oral cancer screening
- **Dental exam:** Inspect teeth for caries, discoloration, fracture, and defective restorations



Radiographs do not determine status of the pulp as pathologic vital pulps are usually not visible on radiographs. Necrotic pulps may not produce radiographic changes in early stages.



According to SLOB rule, the object farthest from the film (most buccal) moves in the direction the cone is directed. Think about why this statement is true.

RADIOGRAPHIC EXAM

- **Periradicular:**
 - Periradicular lesions of pulpal origin show loss of lamina dura apically.
 - The inflammatory process must spread to cortical bone to be visible.
 - If a radiolucency is associated with a vital pulp, it cannot be of pulpal origin and will be either a normal structure or another type of pathosis.
- **Pulpal:**
 - Internal resorption or extensive diffuse calcification may indicate long-term, low-grade irritation.
 - Pulp canal obliteration (calcified canal) does not, in itself, indicate need for treatment.
- **SLOB (same lingual, opposite buccal) rule:**
 - SLOB rule is used to distinguish the lingual from the buccal object on the X-ray film.
 - If the X-ray was shot from the **mesial** side of the tooth, directed toward the distal, the root that appears **mesial** on the X-ray film is the **lingual** root, whereas the buccal root appears on the opposite (distal) side (hence the rule—same lingual, opposite buccal).

DIAGNOSTIC TESTS

Reproduction of chief complaints during diagnostic tests is desirable, if not mandatory.

- **Percussion test:** Determines the presence of inflammation in the PDL.
- **Palpation test:** Indicates the spread of inflammation from the PDL to the overlying periodontium.

- **Periodontal probing:**
 - Provides information with regard to possible etiology and prognosis.
 - Narrow pocket may indicate root fracture or pathosis of pulpal origin.
- **Pulp tests:**
 - Thermal tests:
 - Cold test can be performed using air blasts, ice stick, ethyl chloride, “Endo Ice” (difluorodichloromethane), or CO₂ stick.
 - Hot test can be performed using heated gutta-percha or hot water.
 - Electrical pulp test (EPT):
 - Use of the EPT:
 - Dry off the teeth to be tested.
 - Place some conductive medium (i.e., toothpaste) on the tip of the tester, and have the patient hold onto the handle portion of the tester.
 - Guide the tip of the tester to touch the surface of the tooth.
 - Have the patient let go of the handle when he/she feels the current.
 - EPT is a pulp test that utilizes an electrical current to stimulate sensory nerves of dental pulp.
 - EPT only suggests whether the tooth is vital (= EPT reading of 1–79) or necrotic (= EPT reading of 80).
 - EPT yields high incidence of false-positive and false-negative results.



EPT alone is not sufficient to allow a diagnosis of the pulp and must be combined with other tests.

Diagnosis

- **Pulpal diagnosis:** Pulpal diagnosis is generally achieved by thermal tests (see Table 7–1).
- **Periapical diagnosis:** Periapical diagnosis, as well as pulpal diagnosis, should be formulated for each tooth for which endodontic treatment is planned (see Table 7–2).



No response to thermal tests generally suggests necrotic pulp; however, it may also indicate pulp canal obliteration, immature tooth, recent trauma, previous pulpotomy, premedication, or partially necrotic pulp.

TOOTH CRACKS

CRACKED TOOTH

The crack is noted primarily extending from mesial to distal direction in minimally restored posterior teeth, most commonly in mandibular molars.

TABLE 7–1. Pulpal Diagnosis Based on Thermal Testing

PULPAL DIAGNOSIS	RESPONSE TO THERMAL TESTING
Normal pulp	Mild to moderate pain lasting 1–2 sec after removal of stimulus
Reversible pulpitis	Severe, momentary pain lasting 1–2 sec after removal of stimulus
Irreversible pulpitis	Moderate to severe pain that lingers
Necrotic	No response to thermal tests

TABLE 7 – 2 . Periapical Diagnosis Based on Signs and Symptoms

PERIAPICAL DIAGNOSIS	SIGNS AND SYMPTOMS
Normal periodontium	No periradicular radiolucency and normal response to percussion and palpation.
Acute apical periodontitis	Painful response to biting and percussion.
Chronic apical periodontitis	Periradicular radiolucency without clinical symptoms.
Acute exacerbation of chronic apical periodontitis	Painful response to biting and percussion with periradicular radiolucency.
Acute apical abscess	Rapid onset, spontaneous pain, tenderness to percussion, pus formation, and eventual swelling of associated tissues.
Chronic apical abscess	Gradual onset, little or no discomfort and the intermittent discharge of pus through an associated sinus tract.

- **Symptoms:** Pain to chewing and thermal stimulus is common.
- **Diagnostic tests:**
 - Visual examination of cracks: Aided by staining with a dye, such as methylene blue.
 - Tactile examination: Scratch the tooth surface with a sharp explorer. Widening a gap of the crack may elicit extremely painful response.
 - “**Tooth slooth**” bite tests: Each cusp tip must be tested individually. Pain on release often indicates cracked tooth.
 - **Transillumination:** A crack blocks and reflects the light when a fiberoptic light source is held perpendicular to the plane of the suspected crack.

SPLIT TOOTH

The crack may split the tooth completely into two separate segments. Most often, the split tooth is the result of long-term progression of a cracked tooth.

VERTICAL ROOT FRACTURE

Characterized by a crack that begins in the root and extends toward the occlusal surface, usually in buccal-lingual direction. Endodontically treated teeth are most susceptible.

- **Symptoms:** Patient may report history of abscess or sinus tract formation, as well as pain upon biting or palpation.
- **Diagnostic tests:**
 - Visual verification of cracks.
 - Periodontal probing: A narrow periodontal pocket may form.
 - Radiographs:
 - A diffuse radiolucency along the length of root, especially one with an elliptical or J-shaped appearance may be observed.
 - The fracture line may be observed directly if radiographs are taken from multiple angles.
 - Sinus tract often does not trace to the apex of the root.

Vital Pulp Therapy

INDIRECT PULP CAP

Indirect pulp cap is the application of a dental material over a thin layer of remaining carious dentin after deep excavation with no pulpal exposure.

- **Indications for treatment:** Indirect pulp capping is indicated on permanent teeth with **immature apices** if *all* of the following conditions exist.
 - Tooth has a deep carious lesion that is likely to result in pulp exposure.
 - No history of subjective pretreatment symptoms.
 - No periradicular pathosis on pretreatment radiographs.
- **Procedure:** Treatment consists of two visits approximately 6–8 months apart.
 - First visit: Caries is excavated leaving affected dentin adjacent to the pulp. A thin layer of calcium hydroxide or mineral trioxide aggregate (MTA) is placed over the dentin and the tooth is temporarily restored.
 - Second visit: The restorative material and residual caries mass is removed and the tooth is permanently restored.



The objectives of pulp capping whether direct or indirect, are the formation of reparative dentin and maintenance of a vital pulp.



The reason pulp capping is not used on primary teeth is the alkaline pH of calcium hydroxide or MTA, which can irritate the pulp and induce internal resorption. In permanent teeth, high pH induces reparative dentin formation.



Primary teeth with insufficient root structure (<2/3 of remaining root length), internal resorption, furcation perforation, sinus tract or periradicular pathosis that may jeopardize the permanent successor are not indicated for pulpotomy.

DIRECT PULP CAP

A dental material, such as calcium hydroxide or MTA, is placed directly on a mechanical or traumatic vital pulp exposure.

- **Indications for treatment:** Direct pulp cap is indicated on permanent teeth when *all* of the following conditions exist.
 - Pinpoint mechanical exposure of a clinically vital and asymptomatic pulp.
 - Hemorrhage is easily controlled at the exposure site.
 - Exposure permits the capping material to make direct contact with the vital pulp tissue.
- **Procedure:** A thin layer of calcium hydroxide or MTA is placed directly onto the surface of vital pulp tissue at the site of the pulp exposure followed by a base, and the final restoration is placed over the base.

PULPOTOMY

Pulpotomy is the surgical removal of the coronal portion of a **vital pulp** in an attempt to preserve the vitality of the remaining radicular portion.

- **Indications for treatment:** Pulpotomy is indicated if *any* of the following conditions exist.
 - Exposed vital pulps or irreversible pulpitis of *primary* teeth.
 - As an emergency procedure in *permanent* teeth, for temporary relief of symptoms, until root canal treatment can be accomplished.
 - As an interim procedure for *permanent* teeth with immature root formation to allow continued physiologic root development (apexogenesis).
- **Procedure:** After removal of the coronal portion of the vital pulp tissue, obtain hemostasis and place a biologically acceptable material in the pulp chamber. Two-appointment pulpotomy is indicated if hemostasis could not be achieved in primary teeth.



Pulpotomy procedures in permanent teeth often result in pulp canal obliteration, internal resorption, or necrosis of the remaining root canal system. The canals of these teeth may be inoperable or the root canal therapy may yield poorer prognosis. Therefore, a pulpotomy is regarded as a temporary treatment.

Nonsurgical Endodontics

PRIMARY TEETH

- **Indications for treatment:** Nonsurgical root canal treatment for primary teeth is indicated if *any* of the following conditions exist.
 - Irreversible pulpitis or pulpal necrosis with no evidence of a permanent successor tooth.
 - Pulpal necrosis with or without evidence of periradicular disease.
 - Treatment will not jeopardize the permanent successor.
- **Procedure:** Debridement, enlargement, disinfection, and obturation of all canals are accomplished using an aseptic technique.
 - When a permanent successor tooth is present, the root canal system is obturated with a resorbable root-filling material, such as ZOE.
 - When no permanent successor tooth is present, the canals are obturated with a biologically acceptable nonresorbable material, such as gutta-percha. Root canal sealers are used in conjunction to establish adequate seal.

PERMANENT TEETH

- **Indications for treatment:** Nonsurgical root canal treatment for permanent teeth is indicated if *any* of the following conditions exist.
 - Irreversible pulpitis.
 - Necrotic pulp with or without evidence of periradicular disease.
 - Teeth with a pulp that would be compromised during dental procedures, including denture abutments, malposed teeth, post insertion, and root resection.
 - Cracked or fractured teeth with pulpal involvement.
 - Teeth with thermal hypersensitivity that significantly interferes with normal function, when alternative methods have failed to reduce the hypersensitivity.
- **Procedure:**
 - Proper access is dictated by the size, shape, and location of the pulp chamber and its canal orifices. The entire root of the pulp chamber is removed.
 - Cleaning, shaping, disinfection, and obturation are accomplished using an aseptic technique with dental dam isolation.
 - Following root canal treatment, the tooth must be permanently restored as soon as possible in order to prevent coronal leakage in the root canal system or fracture of the remaining tooth structure.

PROCEDURAL COMPLICATIONS

- **Ledge:** Inadequate use of irrigant and lack of attention to preparation of a glide path or recapitulation lead to a build up of debris within the canal. The file, having lost its natural passage to the working length, will then create a ledge, an artificial irregularity created on the surface of the root canal wall. Once created, ledges are very difficult to bypass.
- **Transportation:** Removal of canal wall structure on the outside curve in the apical half of the canal due to the tendency of files to restore themselves to their original linear shape during canal preparation. Transportation may lead to ledge formation and possible perforation.

- **Perforation:** The mechanical or pathologic communication between the root canal system and the external tooth surface. Root canal treatment, postspace preparation, or root resorption may lead to perforation.
 - Location of perforation, time interval between perforation and repair, and a type of repair material are major factors that affect treatment prognosis.
 - Furcation or cervical perforation, delayed repair time, or extrusion of certain repair materials may result in a poor prognosis.
 - Modern perforation repair materials such as MTA appear to offer a favorable prognosis in the absence of infection.
- **Instrument separation:** It is recognized that intracanal instruments will occasionally separate. Therefore, the practitioner must use sterilized instruments made of biocompatible materials. In the event that the separated fragment cannot be removed or bypassed, the remainder of the accessible root canal space should be obturated with a biocompatible semisolid or solid material. The patient should be informed of any incidence of instrument separation. It is worth mentioning that the separated instrument, per se, does not lead to a failure of endodontic therapy. A separated instrument, however, may lead to a treatment failure if it obstructed proper debridement of the root canal space.
- **Vertical root fracture (VRF):** Characterized by a crack that begins in the root and extends toward the occlusal surface, usually in buccal-lingual direction. VRF necessitates extraction of the tooth. Signs and diagnostic tests were mentioned earlier. Etiology of VRF includes the following iatrogenic errors.
 - Excessive instrumentation.
 - Excess force during compaction of root filling material.
 - Widening of canal during postspace preparation.
 - Unfavorable post length.

Surgical Endodontics

- **Root-end resection (apicoectomy):** The preparation of a flat surface by the excision of the apical portion of the root and any subsequent removal of attached soft tissues.
 - **Indications for treatment:** A root-end resection is indicated if *any* of the following conditions exist.
 - Persistent periradicular pathosis following endodontic treatment.
 - A periradicular lesion that enlarges after endodontic treatment.
 - A marked over-extension of obturation material interfering with healing.
 - When the apical portion of the root with periradicular pathosis cannot be cleaned, shaped, and obturated.
 - **Procedure:**
 - A mucoperiosteal flap is elevated and, when necessary, bone is removed to allow direct visualization of and access to the affected area.
 - Thorough removal of all targeted tissues and/or foreign material is performed.
 - Following root-end resection, a root-end preparation is made and a biocompatible root-end filling material, such as MTA, is placed.
 - Guided tissue regeneration techniques and/or bone replacement may be used prior to closure of the surgical site.



Cervical resorption, once set, is rapid and invasive, and it may necessitate extraction of the involved tooth.

Intracoronar bleaching procedures should be performed with caution.

- **Hemisection:** Hemisection is the separation of a multirooted tooth through the furcation in such a way that a root and the associated portion of the crown may be removed.
- **Root resection (root amputation):** Root resection is the removal of an entire root, leaving the crown of the tooth intact.

► ADJUNCTIVE ENDODONTIC THERAPY

Intracoronar Bleaching

Intracoronar bleaching, also known as nonvital or internal bleaching, is the removal of tooth discoloration using chemical oxidizing agents within the coronal portion of an endodontically treated tooth. Tooth discoloration is often caused by traumatic injury and subsequent pulpal necrosis.

- **Indications for treatment:** An intracoronar bleaching procedure is indicated for a tooth that has *both* of the following clinical conditions.
 - The tooth is discolored from an internal source.
 - Acceptable root canal treatment has been performed if possible.
- **Procedure:**
 - To reduce the potential for cervical resorption, a cement barrier must be placed to minimize the penetration of the bleaching agent into dentinal tubules in the cervical area.
 - After dental dam isolation, coronal portion of the root-filling material is removed to create a room for a cement barrier.
 - A cement barrier, such as IRM or Cavit, is placed in the cervical area.
 - The bleaching agent, typically sodium perborate mixed with saline, is placed in the facial surface of access cavity followed by temporary restoration.
 - The tooth is monitored for a color change every 3–4 days, and the bleaching agent is removed when the satisfactory result is obtained.

Apexification

Apexification is a method to create an apical barrier in a necrotic tooth with an open apex.

- One method of apexification is to induce a calcified apical barrier or to encourage the continued apical development by placing dense calcium hydroxide paste in the root canal space after the instrumentation. The root canals are obturated when the calcified apical barrier is formed in 3–6 months.
- Another method of apexification involves the placement of an artificial apical barrier, such as MTA, prior to obturation. This method, as the root canal treatment can be completed in a day or two, may be appropriate when patient compliance or long-term follow-up care is questionable.

Apexogenesis

Apexogenesis is a vital pulp therapy procedure performed to allow continued physiologic development and formation of the root.

- **Procedure:**
 - Coronal access and pulp amputation.
 - Control hemorrhage.
 - Place calcium hydroxide over the radicular pulp stump.

- Place coronal filling.
- Recall every 3 months to check for the pulpal status, presence of the dentinal bridge, and the continued apical development.
- Root canal treatment is indicated when the root development is completed.

► ROOT CANAL ANATOMY

Access Cavities and Pulp Space Anatomy

- **Access cavity preparation:** Different teeth require different access preparations. Table 7–3 lists the outline access required for each tooth as well as

TABLE 7–3. Outline of Access Cavity and the Number of Canal Openings

Tooth	Outline of Access Cavity	Number of Orifices (Approximate %)			
		1	2	3	4
Maxillary					
Central incisor	Triangle	100			
Lateral incisor	Ovoid	100			
Canine	Ovoid	100			
First premolar	Ovoid	20	75	5	
Second premolar	Ovoid	75	25		
First molar	Triangle			40	60
Second molar	Triangle			60	40
Mandibular					
Central incisor	Ovoid	95	5		
Lateral incisor	Ovoid	70	30		
Canine	Ovoid	95	5		
First premolar	Ovoid	80	20		
Second premolar	Ovoid	90	10		
First molar	Trapezoid			70	30
Second molar	Trapezoid	5 (C-shaped)		90	5



The root canal instrumentation and obturation should stop about 1.0 mm short of the radiographic apex as the apical constriction is positioned 0.5 mm from the apical foramen which is located another 0.5 mm from the radiographic apex.

the typical number of orifices encountered in the pulp chamber. The access cavity preparation must meet the following requirements:

- Allow removal of the entire pulp chamber content.
- Provide direct vision of the chamber floor and canal orifices.
- Facilitate the introduction of instruments into the root canals.
- Provide straight line access to the apical one-third of the canals.
- **Variations in pulp space anatomy:** It is important to remember that there are many variations in the root canal configurations. There may be multiple root canals branching from a single orifice. Conversely, root canals from two separate orifices may join and exit the root apex as a single canal.
- **Location of apical foramina:**
 - **Anatomic apex:** The most apical end of the root. Also known as the radiographic apex.
 - **Apical foramen:** Rarely coincides with the anatomic apex. The mean distance between anatomic apex and apical foramen is about 0.5 mm.
 - **Apical constriction:** Located about 0.5 mm from the apical foramen in the region of the dentin-cementum junction. This should be a natural stop in root canal treatment. This position can be reliably detected by an apex locator.

► ENDODONTIC INSTRUMENTS AND MATERIALS

Hand and Rotary Instruments

- **Hand instruments:**
 - **Broach:** A thin, flexible, fragile, usually tapered and pointed metal instrument with sharp projections or barbs along the length of an instrument. Primarily used to remove pulp tissue from wide root canals.
 - **Reamer:** Manufactured by twisting a tapered, triangular or square wire to form an instrument with sharp cutting edges. Used with a reaming (half-turn twist and pull) action to enlarge root canals. Differs from a K-type file in having fewer spirals or flutes per unit length of cutting blade.
 - **K-file:** K-file has tightly spiraled cutting edges that cut either in reaming or a push-and-pull filing motion. Cross-sectional configurations include diamond, square, and triangular.
 - **Hedstrom file:** Manufactured by cutting the spiral flutes into the shaft of a tapered wire to produce elevated cutting edges that appear to form a series of intersecting cones. Cutting occurs only on a pulling stroke. A Hedstrom file cuts more aggressively than a K-file, but it is more prone to breakage.
- **Rotary instruments:**
 - **NiTi file:** Manufactured by cutting the spiral flutes into a round wire composed of superelastic nickel-titanium alloy. NiTi rotary files remain better centered, produce less transportation, and instrument faster than stainless steel files due to their superior flexibility and resistance to torsional fracture. Hand instruments made of nickel-titanium alloy are also available.

- **Gates-Glidden bur:** Composed of a slender stainless steel shank with a cutting bulb and a pilot-tip. Designed so that a fracture occurs near the hub rather than between the shank and the cutting bulb. The diameter of cutting bulbs ranges from 0.5 to 1.5 mm, corresponding to ISO sizes 50–150.

Intracanal Medication

- **Irrigating solutions:**
 - **Sodium hypochlorite (NaOCl):** A clear, pale, greenish-yellow, strongly alkaline liquid with a chlorine odor. Has a solvent action on **organic** tissue and debris and is a potent antimicrobial agent. NaOCl, used clinically in concentrations from 0.5 to 5.25%, is the current irrigating solution of choice. However, NaOCl bleaches clothing if spilled, and it may cause severe adverse symptoms and reactions if extruded beyond the apex.
 - **Chlorhexidine (CHX):** A bis-biguanide antiseptic agent is used to kill or inhibit microorganisms. CHX is less toxic to our tissue than NaOCl, but it lacks a solvent action. Peridex is a brand name of 0.12% CHX.
- **Chelating agent:**
 - **Ethylene-diamine-tetra-acetate (EDTA):** The aqueous solution removes **inorganic** ions such as calcium to demineralize and soften dentin. This facilitates negotiation and enlargement of calcified canals. EDTA is also used to remove the smear layer in root canals before obturation. EDTA is the active ingredient in RC-PREP, which is often used as a lubricant during instrumentation.
- **Inter-appointment dressing:**
 - **Calcium hydroxide:** An odorless, basic, white powder is an inter-appointment dressing of choice when applied as a creamy suspension in sterile water.
 - Its primary function is antibacterial with added benefits from its cauterizing activity and high pH.
 - The paste consistency also physically restricts bacterial colonization of the canal space.
 - Calcium hydroxide also encourages calcification and inhibits resorption process. Therefore, it is also used in pulp capping, partial pulpotomy, apexogenesis, apexification, and prevention or treatment of root resorption.

Root Canal Filling Materials

Gutta-percha: A gutta-percha point, used to obturate root canals in conjunction with sealers, is a pliable and radiopaque cone available in various sizes. Gutta-percha is pliable at room temperature and becomes plastic at 140°F. Gutta-percha can be dissolved in solvents such as chloroform, xylol, and eucalyptol. Chloroform is often used to remove existing gutta-percha during retreatment root canal therapy.

Sealers: Root canal sealers are used to fill the discrepancies between the canal walls and core materials such as gutta-percha points. Sealers in use today can be divided into four groups based on their constituents: zinc-oxide-eugenol sealers, calcium hydroxide sealers, resin sealers, and glass ionomer sealers. The characteristics of an **ideal sealer** are listed below:

- Nonirritating to periapical tissues
- Insoluble in tissue fluids
- Dimensionally stable
- Hermetic sealing ability
- Radiopaque
- Bacteriostatic
- Good adhesion to canal walls
- Nonstaining to dentin
- Readily removable if necessary

► TRAUMATIC INJURIES

Tooth Fracture

- **Crown fracture without pulp exposure (uncomplicated crown fracture):**
 - **Indications for treatment:** Treatment of uncomplicated crown fracture is indicated when *both* of the following clinical conditions exist.
 - The fracture involves enamel and dentin with no pulp exposure.
 - The pulp tests reveal no indication for endodontic treatment.
 - **Procedure:**
 - In addition to restoring the esthetic aspect of the tooth, procedures for treating uncomplicated crown fractures are intended to protect the dentin and the underlying vital pulp.
 - In immature teeth, continued root development may take place.
- **Crown fracture with pulp exposure (complicated crown fracture):**
 - **Indications for treatment:** Treatment of uncomplicated crown fracture is indicated when *both* of the following clinical conditions exist.
 - The fracture involves enamel, dentin, and exposure of the pulp.
 - The pulp is vital.
 - **Procedure:**
 - For immature teeth:
 - The purpose of treatment is to protect the pulp so that root development may continue.
 - Pulp capping or partial pulpotomy procedures are indicated.
 - A biologically acceptable material, such as MTA or calcium hydroxide, is placed directly in contact with the pulp to maintain the vitality and function of the remaining radicular pulp.
 - A final restoration is placed.
 - When the root reaches full maturation, root canal treatment and crown placement may be indicated.
 - For mature teeth:
 - If the fractured tooth does not show signs and symptoms of irreversible pulpitis and a crown restoration is not necessary, it is acceptable to use pulp capping or partial pulpotomy followed by bonded composite resin.
 - In any other case, root canal treatment is indicated.
- **Crown-root fracture:**
 - **Indications for treatment:** Crown-root fracture involves enamel, dentin, and cementum that may or may not involve the pulp.

- **Procedure:**
 - For immature teeth:
 - The purpose of treatment is to protect the pulp so that root development may continue.
 - The same procedures as for complicated crown fracture are indicated.
 - In addition, soft tissue surgery to allow access to the fracture site may be necessary.
 - For mature teeth:
 - Root canal treatment is indicated in most cases.
 - Procedures to facilitate restorations may include, but are not limited to, crown lengthening surgery and orthodontic extrusion.
- **Root fracture:**
 - **Indications for treatment:** Root fracture involves cementum, dentin, and pulp, and may be horizontal or oblique.
 - **Procedure:**
 - Immediate care includes reduction and stabilization of the fracture site.
 - The fractured coronal segment is repositioned into the normal position and immobilized with a flexible splint. The occlusion is relieved.
 - Definitive care is limited to periodic radiographic and clinical evaluations.
 - If pulpal necrosis develops, root canal treatment of the **coronal segment** is indicated.
 - If coronal tooth structure is lost at the level of the crestal bone, root extrusion or crown lengthening surgery may be indicated.

Tooth Displacement

- **Luxation:** Luxation is displacement of a tooth from its original position in the alveolus, without total avulsion, resulting from acute trauma. Luxation can be categorized as concussion, subluxation, extrusive, lateral, and intrusive (see Table 7-4).



Hank's balanced salt solution or milk, due to its physiologic osmolality, provides the best storage condition, followed by saline and saliva. Tap water is equally as damaging as dry storage.

TABLE 7-4. Classification and Clinical Signs of Luxation

TYPE OF LUXATION	CLINICAL SIGNS
Concussion	Trauma resulting in sensitivity to percussion but little or no mobility and no displacement.
Subluxation	Injury to supporting tissues resulting in abnormal loosening of the tooth without displacement.
Extrusive luxation	Partial axial displacement of the tooth out of its socket.
Lateral luxation	Displacement of the tooth in a direction other than axially that can be accompanied by fracture of the alveolar socket.
Intrusive luxation	Axial displacement of the tooth into the alveolus and can be accompanied by fracture of the alveolar socket.

TABLE 7 – 5. Treatment of Luxated Permanent Teeth

TYPE OF LUXATION	TREATMENT
Concussion or subluxation	■ No treatment is needed in majority of cases. ■ A flexible splint can be placed for the patient’s comfort for 7–10 days. ■ Root canal treatment is initiated if the tooth becomes symptomatic or if it shows signs of necrosis, periodontitis, ankylosis, or resorption.
Extrusive luxation or lateral luxation	■ Reposition the tooth into normal position. ■ Stabilize the tooth with a flexible splint for up to 3 weeks. ■ Root canal treatment is initiated if the tooth becomes symptomatic or if it shows signs of necrosis, periodontitis, ankylosis, or resorption.
Intrusive luxation	■ Slightly luxate the tooth with forceps. ■ Spontaneous reposition/reeruption (teeth with incomplete root formation) or orthodontic repositioning (teeth with completed root formation) or surgical repositioning is performed. ■ Perform prophylactic root canal treatment 1–3 weeks after injury.

- **Indications for treatment:** Luxation includes slight to severe injuries to teeth and their supporting structures.
- **Procedure:** Management of injuries differs according to the type of luxation (see Table 7–5).
- **Avulsion:** Avulsion is the complete dislodgment of a tooth out of its socket by traumatic injury. Short **extra-oral dry time** and proper **storage medium** are key factors in offering favorable treatment outcome.
 - **Indications for treatment:** Treatment is indicated when a tooth is completely dislodged from its alveolus.
 - **Procedure:** Stage of root development, extra-oral dry time, and the type of storage medium should be considered (see Tables 7–6 and 7–7).

TABLE 7 – 6. Treatment of Avulsed Teeth with Open Apex

CLINICAL SITUATION	TREATMENT
Extra-oral dry time <60 minutes or the tooth kept in special storage media.	■ Clean the root surface with saline. ■ Irrigate the tooth socket with saline. ■ Gently replant the tooth. ■ Stabilize using a flexible splint for 1 week or until mobility is minimal. ■ Pulpal revascularization may occur, so the tooth should be monitored regularly for evidence of pulpal vitality and continued root development. ■ For teeth in which vitality does not return, apexification procedures are followed by root canal treatment.
Extra-oral dry time >60 minutes	■ Replantation is not indicated.

TABLE 7 – 7. Treatment of Avulsed Teeth with Closed Apex

CLINICAL SITUATION	TREATMENT
Extra-oral dry time <60 minutes or the tooth kept in special storage media	■ Clean the root surface with saline ■ Irrigate the tooth socket ■ Gently replant the tooth ■ Stabilize using a flexible splint for 1 week or until mobility is minimal ■ Root canal treatment is performed at the time of splint removal
Extra-oral dry time >60 minutes	■ Replantation is generally not indicated

Periodontics

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Periodontium

- Gingiva
- Periodontal ligament (PDL)
- Cementum
- Alveolar and supporting bone

Gingiva

- Those parts of the masticatory mucosa which cover the alveolar process and surround the cervical portion of teeth.
- The gingiva is composed of connective tissue and epithelium.
- Epithelium can be divided into three histological distinct areas:
 - Oral epithelium—continuous with epithelial lining of the attached gingiva
 - Composed of keratinized stratified squamous epithelium
 - Sulcular epithelium
 - Nonkeratinized
 - Junctional epithelium
 - Attached to the tooth by hemidesmosomes
 - Nonkeratinized
 - Larger cells with increased intercellular spaces
- **Gingival crevicular fluid (GCF):**
 - Transudate that emerges from the gingival sulcus.
 - Contains a variety of enzymes and cells, particularly desquamating epithelium and neutrophils that are being shed through the sulcus.
 - An increase in gingival crevicular flow is the first detectable sign of inflammation.
 - Once inflammation has occurred, the GCF is referred to as an inflammatory exudate.
 - This exudate contains a higher level of serum proteins and leukocytes.
- **Gingival fibers:**
 - Composed of type I collagen.
 - Support the gingiva and attach it to the tooth and alveolar bone.
 - Also known as supracrestal connective tissue fibers.
 - Designated by their orientation:
 - Dentogingival fibers
 - Dentoperiosteal fibers
 - Circular fibers
 - Alveologingival fibers
 - Transseptal fibers
 - Gingival fibers are continuous with the periodontal ligament.
 - PDL is also considered to be connective tissue.
- **Gingival apparatus:**
 - Gingival fibers and the epithelial attachment
- **Stippling:**
 - Refers to the irregular surface texture of the attached gingival, similar to the surface of an orange peel.
 - Occurs at the intersection of epithelial ridges that causes depression and the interspersing of connective tissue papillae between these intersections giving rise to the small bumps.



Transseptal fibers connect two adjacent teeth.

- All of the following may result in an absence of “stippling” in areas of the attached gingiva:
 - Edema of the underlying connective tissue
 - Inflammatory degradation of gingival collagen fibers
 - Normal variation in gingival topography
- Stippling is usually not visible until age six.
- **Circular fibers:**
 - Encircle the tooth around the most cervical part of the root and insert into the cementum and lamina propria of the free gingiva and the alveolar crest.
 - Resist rotational forces.
- **Transeptal fibers:**
 - Extend from tooth to tooth, coronal to the alveolar crest and are embedded in the cementum of adjacent teeth.
 - Not found on the facial aspect and have no attachment to alveolar crestal bone.
 - Maintain the integrity of the dental arches.
 - Sometimes classified within the principal fibers of the PDL.
- **Dentogingival fibers:**
 - Extend from the cementum apical to the epithelial attachment and course laterally and coronally into the lamina propria of the gingiva.
- **Dentoperiosteal fibers:**
 - Extend from the cervical cementum over the alveolar crest to the periosteum of the cortical plates of bone.
- **Alveologingival fibers:**
 - Insert in crest of alveolar process and spread out through the lamina propria into the free gingiva.
- **Junctional epithelium (JE):**
 - Collar-like band of stratified squamous epithelium 10–29 cells thick near the sulcus and 2–3 cells thick at the apical end.
 - In health, usually is 0.25–1.35 mm long (~1.00 mm).
 - It is the stratified nonkeratinizing epithelium that surrounds the tooth like a collar.
 - Attached by one broad surface to the tooth and by the other to the gingival connective tissue.
 - Has two basal laminae, one that faces the tooth (internal basal lamina) and one that faces the connective tissue (external basal lamina).
 - Proliferative cell layer responsible for most cell divisions located in contact with the connective tissue (i.e., next to the external basal lamina).
 - Desquamative (shedding) surface of the JE is located at its coronal end, which also forms the bottom of the gingival sulcus.
 - JE is more permeable than the oral or sulcular epithelium. Serves as the preferential route for the passage of bacterial products from the sulcus into the connective tissue and for fluid and cells from the connective tissue into the sulcus.
 - In disease, migration of JE occurs, along with the degeneration in connective tissue under the attachment; as the junctional epithelium proliferates along the root surface (gets longer) the coronal portion detaches.
 - Barrier membranes, which are often used to treat bony defects, help to prevent this long junctional epithelium from forming.

- **Epithelial attachment:**
 - Special part of the junctional epithelium that actually provides the attachment.
 - Consists of lamina lucida, lamina densa, and hemidesmosomes.

Principal Fibers of the Periodontal Ligament

- **Horizontal**
- **Alveolar crest fibers:**
 - Extend from the cervical cementum of the tooth to the alveolar crest
 - Function is to counterbalance the occlusal forces on the more apical fibers and resist lateral movements
- **Oblique fibers:**
 - Obliquely situated with insertions in the cementum and extending more apically in the alveolus
 - Fibers are more resistant to forces along the long axis of a tooth (masticatory forces)
 - Most likely to be found in the middle third of the root
 - Most numerous
- **Apical fibers:**
 - Radiate apically from cementum of tooth to bone
 - Offer initial resistance to tooth movement in an occlusal direction
- **Interradicular fibers:**
 - Found only on multirooted teeth
 - Extend from the cementum in the furcation area to the bone within the furcation
- **Alveolar bone proper:**
 - Section of the alveolar process which immediately surrounds the root of the tooth and to which the fibers of the PDL are attached
 - Perforated cribriform plate through which vessels and nerves pass between the PDL and marrow
 - Consists of two layers of bones:
 - Compact lamellar bone
 - Bundle bone
 - Layer into which the periodontal fibers insert
- **Supporting alveolar bone:**
 - Osseous structure which surrounds the alveolar bone proper and gives support to the socket
 - Consist of cortical plate (compact lamellar bone)
 - Forms the outer and inner plates of the alveolar processes
 - Thicker in the mandible than in the maxilla
- **Spongy bone (cancellous bone):**
 - Fills in area between cortical plates of bone.
 - This type of bone is not present in the anterior region of the mouth.
 - In the anterior region, the cortical plate is fused to the cribriform plate.
 - Spongy bone exists over the radicular buccal bone of the maxillary posterior teeth.
- **Periodontal ligament:**
 - Specialized form of connective tissue derived from the dental sac that surrounds the root of a tooth and attaches it to its bony socket.
 - Made up primarily of connective tissue fibers (collagen fibers).
 - Complex, soft, connective tissue containing numerous cells, nerves, blood vessels, and extracellular substances consisting of fibers (gingival and principal) and ground substance.



Principal fibers of the PDL are bundles of collagen fibers grouped according to the direction they extend from the cementum of the root to the alveolar bone.



A dehiscence is the loss of the buccal or lingual bone overlaying the root portion of a tooth, leaving the area covered by soft tissue only.



Orthodontic treatment is possible because the PDL continuously responds and changes as the result of the functional requirements imposed upon it by externally applied forces.



The fibers of the periodontal ligament are arranged into two groups: gingival fibers and principal fibers.



Principal fibers (collagen fibers) of the periodontal ligament include alveolar crest, horizontal, oblique, apical and interradicular fibers.

- The majority of the periodontal fibers are collagen and the ground substance is composed of a variety of proteins and polysaccharides.
- Thickness of PDL varies on person's age, stage of eruption, function of the tooth, history of any trauma.
- Specialized cells of the ligament function to resorb and replace the cementum (cementoclasts or cementoblasts), periodontal ligament (fibroclasts or fibroblasts), and alveolar bone (osteoclasts or osteoblasts).
- The cell type is most frequently found in the periodontal ligament is the fibroblast.
- **Gingival fibers:**
 - Connective tissue fibers that are found in the free gingiva, however, are continuous with the connective tissue fibers of the periodontal ligament and are often considered to be a part of the ligament.
 - Provide support for the marginal gingiva (including the interdental papilla).
 - Include circular, dentogingival, dentoperiosteal, alveologingival and transeptal fibers.
 - Connect the cementum to the alveolar bone.
 - Distinguished by their location and direction.
 - The terminal portions of these collagen fibers, which are embedded into the cementum and alveolar bone, are called Sharpey's fibers.
- **Free gingiva:**
 - Collar of tissue that is not attached to the tooth or alveolar bone
 - Includes gingival margin, free gingival groove, gingival sulcus, and interdental (interproximal) gingiva
- **Gingival margin:**
 - The most coronal portion of the gingiva
- **Free gingival groove:**
 - Demarcation of separation from free gingiva to attached gingiva. Only present in one-third of adults.
- **Gingival sulcus:**
 - Shallow groove between the marginal gingiva and the tooth surface
 - Bound by sulcular epithelium laterally and junctional epithelium apically
- **Interdental (interproximal) gingiva:**
 - The gingiva that occupies the interdental spaces coronal to the alveolar crest
- **Attached gingiva:**
 - Part of the gingiva that is attached to the underlying periosteum of the alveolar bone and to the cementum by connective tissue fibers and the epithelial attachment
 - It is present between the free gingiva and the more movable alveolar mucosa
- **Mucogingival junction:**
 - Separates attached gingiva from the alveolar mucosa

► DIAGNOSTIC

Periodontal Examination

- Bleeding upon probing (BOP).
- Pocket depths.
- Mobility.

- Radiographic evidence of bone loss.
- A change in tissue color and tone.
- Furcation evaluation.
- Plaque and calculus.
- Level of the free gingival margin in relation to the cemento-enamel junction (CEJ):
 - Normal level of epithelial attachment should be on enamel or at the CEJ:
 - This would place the free gingival margin 2–3 mm coronal to the sulcular base.
- Periodontal pocket depth:
 - All measurements are recorded for sulcular depth.
 - Probing depth is the distance in millimeters from the gingival margin to the deepest point reached by the probe.
 - Accuracy of measurement of the pocket depths are dependent upon:
 - Probing force (maximum 25 g)
 - Degree of inflammation in soft tissues
 - In disease one could probe through epithelial attachment and connective tissue all the way to osseous crest
 - Position, angulation, and orientation of probe
 - Presence of calculus
 - Root morphology
 - Restricted access and/or visibility
- The loss of attachment is determined by measuring the distance between CEJ and the base of the attachment.
- Bleeding:
 - Physiologically, bleeding from the gingival sulcus should not be caused by gentle provocation.
 - Bleeding in the absence of local irritants may indicate a systemic disease.
 - When inflammation is caused by plaque accumulation, it causes ulceration in the epithelium.
- Exudate—the presence of exudates, specifically suppuration, is evaluated by digital pressure on the buccal and lingual of each tooth. Exudate cannot be expressed from the gingival sulcus in gingival health.
- Mucogingival complications—the involvement of not only the gingival component in the disease state, but also the presence of imminent involvement of the alveolar mucosa.



Bleeding on probing is one of the major indicators of gingival inflammation.



Pathological mobility is dependent on the quantity of remaining bony support, the degree of inflammation, and the magnitude of occlusal forces. A reduction in mobility can follow treatment and resolution of inflammation.

Tooth Mobility

Tooth mobility is important in the development of a prognosis and vital to treatment planning. Mobility is determined by gently tapping the tooth in a buccal-lingual direction with two instruments.

- Physiological mobility—allows slight movement of tooth to accommodate masticatory forces, without injury to the tooth or its supporting tissues.
- Pathological mobility—increased mobility as a result of connective tissue attachment loss.
- Miller's classification:
 - Grade I—horizontal mobility <1 mm.
 - Grade II—horizontal mobility ≥1 mm.
 - Grade III—horizontal mobility >2 mm and/or vertical mobility.

Glickman Furcation Classification

- Grade I—Incipient bone loss. Furcation probe can feel the depression of the furcation opening.
- Grade II—Partial bone loss. Probe tip enters under the roof of the furcation.
- Grade III—Total bone loss with through-and-through opening of the furcation. The furcation entrance is not visible clinically (see Fig. 8–1A).
- Grade IV—A Grade III furcation where the furcation entrance is visible clinically.
- Furcation treatment depends on the severity of the lesions. Options include:
 - Local delivery antibiotics, scaling and root planning, surgical flap elevation gaining direct access for instrumentation, furcationplasty, bone grafting, guided tissue regeneration, tunnel preparation (see Fig. 8–1A to 8–1D), root resection (see Fig. 8–2A to 8–2E), and extraction



FIGURE 8–1A. Class III furcation on tooth #19—initial visit—Class III furcation not visible clinically. (Also see color insert)

Tunnel Preparation of Class III Furcation (Courtesy of Gaston Berenguer, D.M.D., M.S.)



FIGURE 8–1B. Two month reevaluation of newly created Class IV furcation (periodontal surgery). (Also see color insert)

Tunnel Preparation of Class III Furcation (Courtesy of Gaston Berenguer, D.M.D., M.S.)



FIGURE 8-1C. Furcation now accessible for hygiene with proxibrush. (Also see color insert)

Tunnel Preparation of Class III Furcation (Courtesy of Gaston Berenguer, D.M.D., M.S.)

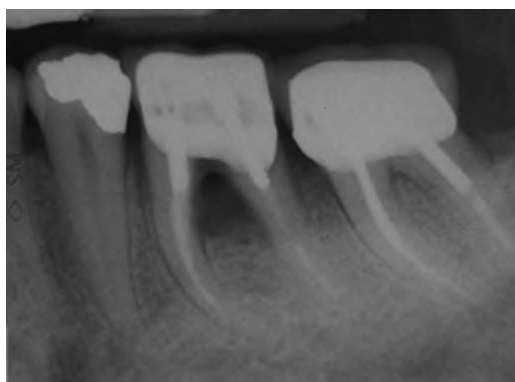


Figure 8-1D. Posttreatment radiograph of tooth #19.

Tunnel Preparation of Class III Furcation (Courtesy of Gaston Berenguer, D.M.D., M.S.)



FIGURE 8-2A. Initial visit—Class III furcation on tooth #31.

Root Resection (Courtesy of Mark Forrest, D.M.D.)

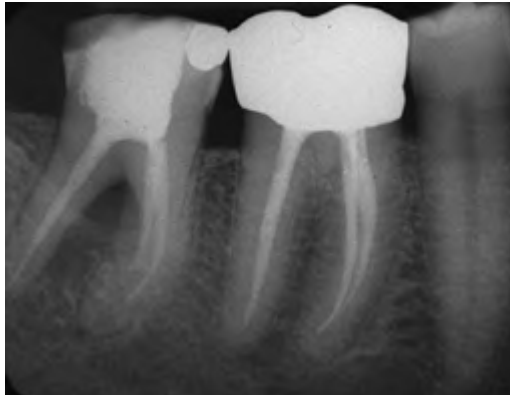


FIGURE 8-2B. Endodontic treatment completed on tooth #31.

Root Resection (Courtesy of Mark Forrest, D.M.D.)



FIGURE 8-2C. Tooth #31 hemisected.

Root Resection (Courtesy of Mark Forrest, D.M.D.)



FIGURE 8-2D. Distal root of tooth #31 extracted (resection).

Root Resection (Courtesy of Mark Forrest, D.M.D.)

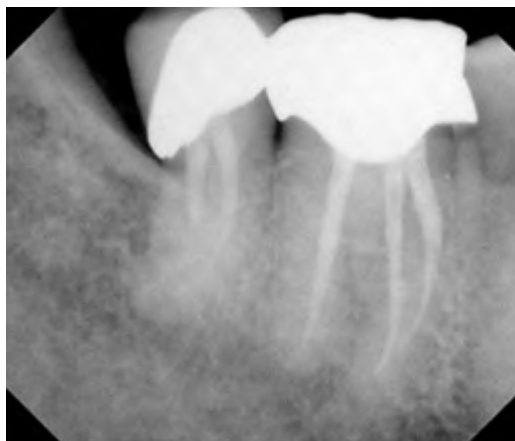


FIGURE 8-2E. Eighteen year reevaluation of tooth #31.

Root Resection (Courtesy of Mark Forrest, D.M.D.)

► PERIODONTAL PATHOLOGY

Tooth Brush Trauma

- Usually occurs on teeth that are the most prominent in the dental arch:
 - Canines
 - Premolars
- Hard-bristle toothbrush is capable of causing gingival recession and abrasion of enamel and/or cementum and should be avoided.
- The abrasive quality of dentifrices affects enamel, but abrasion is more of a concern for patients with exposed dentin.
 - Dentin is abraded 25 times faster and cementum 35 times faster than enamel.
- Trauma from brushing may result in recession of the marginal gingiva, lacerations of the soft tissues including the attached gingiva and the alveolar mucosa, v-shaped notches in the cervical areas of teeth, and gingival clefts.

Gingival Overgrowth

- Most commonly caused by medications such as phenytoin (Dilantin), cyclosporine A, and nifedipine (see Fig. 8-3).
- Appears to be related to the level of plaque accumulation.
 - Dilantin hyperplasia is a progressive proliferation response to the gingiva associated with the use of sodium Dilantin (phenytoin).
 - Recent studies have shown that the administration of this medication does not cause significant increase in fibroblast activity.
- The occurrence of the gingivitis is not automatic with the drug therapy if the level of oral hygiene is maintained and no preexisting gingival disease exists.
- A significant difference in comparing this hyperplasia to hereditary gingivofibromatosis is the finding that there is an increased accumulation of inflammatory cells in dilantin-induced hyperplasia.



Studies have shown that between 50 and 60% of persons taking Dilantin will develop hyperplasia.



Other medications that can cause gingival hyperplasia include calcium channel blockers (verapamil, felodipine, diltazem, amlodipine), antiepileptics, the immunosuppressive mycophenolate, the antidepressant sertraline, the antipsychotic pimozide, and interferon alpha-2-beta.



The best criterion to evaluate the success of scaling and root planning is no evidence of bleeding upon probing since the presence of bleeding indicates active inflammatory periodontal disease.



FIGURE 8-3. Medication induced gingival hyperplasia.

(Courtesy of Andrew Forrest, D.M.D., M.S.)

Pseudopocketing

- Condition where pocketing occurs without attachment loss.
- Also referred to as a gingival or relative pocket since it is caused by expansion of the marginal tissue coronally rather than an apical movement of the epithelial attachment beyond its physiological level.
- Classic example of pseudopocketing is found with Dilantin hyperplasia.

► THERAPY

Scaling and Root Planing

- Techniques of instrumentation applied to the root surface to remove it of plaque, calcified deposits, and softened or roughened cementum.
- When thoroughly performed, these techniques provide a smooth, clean, hard polished root surface.
- Primary treatment for periodontal inflammation.
 - In simple cases, this treatment is useful in reducing shallow pockets and reducing the number of bacteria within these shallow pockets and may be the only treatment necessary.
- In severely advanced periodontal disease where surgery may not be possible, scaling and root planning are the only treatment feasible.
- Since the removal of plaque and deposits is the most definitive treatment for periodontal inflammation, root planning and scaling are the more frequently used than any other type of therapy.
- Commonly observed clinical changes 1 week after scaling and root planning include a reduction in pocket depths and a reduction in gingival inflammation (see Fig. 8-4A and 8-4B).

Periodontal Curette

- A narrow, delicate instrument having either one or two cutting edges or working surfaces.
- The end of the edge is rounded.
- Most often curettes are in paired forms allowing for access to opposite surfaces.



FIGURE 8-4A. Initial visit.

Scaling and Root Planing (Courtesy of Andrew Forrest, D.M.D., M.S.)

- They are generally smaller than scalers and are designed to permit atraumatic entry to the subgingival space.
- Tactile sensitivity of most curettes is greater than scalers and is well suited for subgingival calculus detection, calculus removal, and root planning.
- Factors to be considered in instrumentation selection for root planning include:
 - Location of the calculus deposit
 - Area of the mouth to be instrumented
 - Adaptation of gingiva to the tooth
- Using curettes with short, even working strokes followed by longer ones is the most effective and efficient method of performing root planning.
- Final root planning strokes are longer and lighter than scaling strokes.
- Root planning strokes become lighter as the cementum becomes smoother.
- Exploratory scaling and root planning strokes differ in angulation, pressure, length, and direction.



FIGURE 8-4B. Scaling and root planing with ultrasonics and hand instruments—6 week reevaluation.

Scaling and Root Planing (Courtesy of Andrew Forrest, D.M.D., M.S.)



Trauma from occlusion is reversible as long as the insult can be eliminated and the body can repair the damage.

Trauma from Occlusion

- Radiographic signs of trauma from occlusion:
 - Widening of periodontal ligament space
 - Possible thickening of lamina dura
 - Angular bone loss and infrabony pocket formation
 - Root resorption
 - Hypercementosis
- Periodontal pockets are not caused by occlusal trauma.
- Occlusion has little significance in the etiology of periodontal disease, but it may act as a contributing factor.
- A local irritant and inflammation are necessary to cause apical shift of the attachment.
- Primary occlusal trauma refers to excessive force applied to a tooth or teeth with normal supporting structures.
- Secondary occlusal trauma refers to excessive force applied to a tooth or teeth with inadequate support (periodontal disease).

Findings Associated with Excessive Occlusal Forces

- Alternating areas of resorption and repair of the alveolar bone
- Fibrosis
- Cemental resorption leading to dentinal resorption
- Cemental tears
- Possible ankylosis
- Occlusal pulpal necrosis and calcification
- Radiographic changes that may be seen on teeth that are no longer in function:
 - Reduced trabeculation of bone
 - Narrowing of the periodontal ligament space

Selective Grinding

- Achieve a more favorable distribution and direction of forces in the natural dentition.
- Coordinate the median occlusal position with the terminal hinge position of the mandible.
- Eliminate prematurities in excursive movements to gain canine or group function.
- Direct forces along the long axis of tooth.
- Establish or maintain masticatory function.
- Accomplish occlusal adjustment without adjusting vertical dimension and leaving adequate amount of interocclusal distance.
- Reduce or eliminate fremitus.
- Eliminate prematurities first in centric relation, then protrusive movements, followed by lateral excursive movements.

Bruxism

- Aggressive, repetitive and continuous grinding, gritting or clenching of the teeth during the day/night in other than functional activities such as chewing or swallowing

Primary Occlusal Trauma

- Condition when pathological occlusal forces exceed the normal capacity of a normal periodontium causing damage to the attachment apparatus.
- Usually reversible once the forces are controlled.
- Early effect of trauma includes hemorrhage and thrombosis of blood vessels in the PDL.

Secondary Occlusal Trauma

- Normal occlusal forces are subjected on a reduced periodontium leading to further inflammation and bone loss.
- Mobility is a common clinical sign of occlusal trauma.

Maintenance

- Frequency of recalls is determined by the periodontist.
- The clinical condition and appearance of the periodontium and the patient's ability to perform adequate home care will govern the frequency.
- Important to motivate and reinforce good habits for patient.
- First year after treatment is critical since patient has already demonstrated susceptibility to periodontal disease.
- Dental implants have different instruments associated with their maintenance. The instruments are usually plastic-tipped so that the surface of the implant is not scratched.

► GINGIVITIS AND PERIODONTAL DISEASE

Gingivitis

- The form of periodontal disease that is associated with plaque.
- Resolves completely when plaque is removed.
- Localized protective response elicited by bacterial invasion.
 - Serves to destroy, dilute, or wall off both the injurious agent and the injured tissue.
 - Indirect mechanisms (host response) combine with direct mechanism (bacteria and their products) to determine the intensity of the inflammatory reaction.
- **No loss of attachment.**
- **No bone loss.**



The etiology of gingivitis is microbial plaque.

Clinical Criteria Used for Diagnosing Gingivitis

- Color:
 - Normal healthy gingival color (Fig. 8–5A) ranges from coral pink to various stages of pigmentation.
 - Erythema refers to an intense red color (Fig. 8–5B and 8–5C).
 - Probably the most common color change noted with periodontal disease is cyanosis (bluish-purple hue).



FIGURE 8-5A. Healthy gingiva. (Also see color insert)

Healthy Gingiva (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-5B. Gingivitis. (Also see color insert)

Gingivitis (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-5C. Gingivitis with plaque disclosing solution (erythrosine). (Also see color insert)

Gingivitis (Courtesy of Andrew Forrest, D.M.D., M.S.)

- Contour:
 - Has range of normal.
 - Influenced by missing teeth, position of teeth, and so forth.
 - Papillae should fill the interproximal spaces.
 - Gingival margins should be scalloped in form.
 - Gingiva should be firmly attached to the teeth.
- Tone/consistency of the gingival tissue
 - Should be resilient and fibrotic in nature from the free gingival groove, apical to the mucogingival junction.
 - Texture.
 - Stippling of the attached gingiva should be present.

Three Stages of Disease in Developing Gingivitis

- *Transient (incipient) stage*—within 2–4 days after cessation of oral hygiene, the earliest changes are visible microscopically. These consist of a margination of leukocytes (polymorphonuclear neutrophils [PMNs]) in vessels close to the junctional epithelium. Sloughed epithelial cells and bacteria are found in the gingival sulcus.
- *Developing stage*—the area of collagen destruction becomes larger and is occupied by fluid that contains serum proteins: fibrin; immunoglobulins, (especially IgG); complement; inflammatory cells (B or T lymphocytes, macrophages).
- *Chronic stage*—the cytologic characteristics of the inflammatory infiltrate in the gingival lamina propria are changed.
- IgG is produced by most of the plasma cells.
- A few cells containing IgA are present (mostly in saliva).
- IgM-containing cells are rarely seen.

Cytokines

- Immune system produced infection-fighting factors that are involved in healing.
- In excess, they can cause inflammation and severe damage.
- Some can cause an overproduction of collagenase which breaks down proteins, including the protein-containing connective tissues that support the teeth.
- Patients with abnormally hyper-responsive inflammatory reactions often have a hyper-inflammatory monocyte/macrophage phenotype.

B-Cells

- White blood cells that complete maturation in bone marrow and migrate to lymphoid organs.
- Committed to differentiate into antibody-producing plasma cells involved in antibody-mediated immunity.
- When an immature B-cell is exposed to a specific antigen, cell is activated.
- It then travels to the spleen or lymph nodes and differentiates, and rapidly produces plasma cells and memory cells.
- Plasma cells synthesize antibodies.



The size of healthy gingival tissues should be well contoured to the underlying osseous architecture with the free gingival margin being of such thinness to allow for a “knife edge” thickness at the dentogingival margin.



Lymphocytes are the predominant cell of the developing stage of gingivitis.



Plasma cells are the predominant cell of the chronic stage of gingivitis.



Studies have suggested that this inflammatory response may have damaging effects not only on the connective tissue that supports the teeth but also in organs throughout the body, including the heart.

T-Cells

- White blood cells that mature in the thymus and become thymocytes
- Important in cell-mediated immunity, in type IV hypersensitivity reactions (contact dermatitis), and in the modulation of antibody-mediated immunity
- Major classes include helper T-cells, suppressor T-cells, and cytotoxic T-cells

Neutropenia

- Abnormal decrease in the number of neutrophils in the blood
- Associated with acute leukemia, infection, rheumatoid arthritis, vitamin B12 deficiency, and chronic splenomegaly

Leukemias

- Malignant neoplasms of immature white blood cells
- Patients with acute leukemias have more oral complications than those with chronic leukemias
- Gingiva is grossly enlarged and is bluish-red in color:
 - Soft, spongy consistency
 - Blunted papilla

► INFLAMMATION

Initial Lesion of Gingivitis

- PMNs are first to appear in the gingival sulcus due to inflammation caused by plaque formation and migrate along chemical gradients toward the injured site.

Early Lesion of Gingivitis

- Occurs around 4–7 days. Area of destruction becomes larger with persistence of inflammation.
- Leukocytes invade connective tissue and are dominated by lymphocytes (3/4 of all cells), macrophages, plasma cells (secrete IgG), and mast cells (release histamine).

Chronic Lesion of Gingivitis

- The time varies. There is an increase in plasma cells (still secreting IgG) and B-lymphocytes. Invade deep into connective tissue. This stage may persist for years with or without going to the advanced lesion or periodontitis.



Neutrophils are the most numerous cells in the inflammatory exudates of an acute periodontal abscess.

Advanced Lesion of Gingivitis

- Advanced lesion—lymphocytes, plasma cells, and macrophages continue to invade the connective tissue along vascular pathways and destroy the gingival fibers. Changes within the supportive bone occur as the inflammatory process continues.

Pregnancy Gingivitis

- Generalized, marginal, and edematous gingivitis.
- Gingival bleeding is the most common complaint.
- Severity tends to increase from the second to the eighth month of pregnancy.
- Often some resolution during final trimester and after parturition.
- A local gingival overgrowth (epulis) may result from chronic irritation or mild trauma to the soft tissues.
- The etiology is an increase in circulating levels of estrogen, progesterone, and their metabolites.
 - The hormones cause an increase in gingival vasculature and the permeability of the capillary network.
 - A similar increase in the severity of gingivitis may also be seen at or around puberty and with long-term use of oral contraceptives.
- Treatment includes oral hygiene instructions and scaling.

Four Local Signs of Acute Inflammation

- Redness (rubor):
 - Due to dilation of capillaries
- Heat (calor):
 - Characterized by dilation of blood vessel leading to increased blood flow and high metabolic rate of neutrophils and macrophages
- Swelling (tumor):
 - From increased cellular permeability
- Pain (dolor):
 - Due to lysis of cells triggers production of bradykinin and prostaglandins

Mast Cells

- Found in connective tissue.
- Contain numerous basophilic granules and release heparin and histamine due to injury or inflammation.
- Mast cell histamine is released when tissues are damaged.
- Histamine is important in the vascular phase of acute inflammation (causing vasodilation and increased vascular permeability).
- Mast cell content in human gingiva is high.
 - Mast cell content of inflamed gingiva increases as the severity of inflammation increases.



Histamine is stored in mast cells, platelets, and basophils.

Two Phases of Acute Inflammation

VASCULAR PHASE

- Initially starts with vasoconstriction (temporary) due to narrowing of blood vessels caused by contraction of smooth muscle in the vessel walls
 - Can be seen as blanching of the skin
- Followed by vasodilation (widening of blood vessels) to increase the blood flow to the infected area
- Leads to increased vascular permeability which allows diffusible components to enter the site



Cells involved in acute inflammation include basophils, tissue mast cells, and platelets.



Periodontitis is the inflammation of the supporting tissues of the teeth. It is usually a progressive destructive change leading to bone loss and breakdown of the periodontal ligament.



In periodontitis there is an apical migration of the junctional epithelium and the formation of a long junctional epithelium.

CELLULAR PHASE

- Leukocytes (predominantly PMNs) are the first line of defense cells to migrate to the injured tissue by chemotaxis and ingest particulate matter by phagocytosis.
- Engulfed matter becomes a phagosome and then combines with lysosomal granules to form a phagolysosome, in which digestion of the engulfed particle occurs.
- Macrophages appear late in the cellular phase and represent a transition between acute and chronic inflammation.

► PERIODONTITIS

Periodontitis

- Inflammation that affects and destroys the attachment apparatus.
- Histology is marked by apical migration of the junctional epithelium from the CEJ, loss of connective tissue attachment, loss of periodontal ligament, and destruction of bone.
- Generally progresses slowly and painlessly and can be arrested with proper therapy.

Histopathological features of periodontitis:

- Predominance of plasma cells.
- Continuing loss of collagen subadjacent to the pocket epithelium with fibrosis at more distant sites.
- Formation of periodontal pockets.
- Extension of lesion into alveolar bone and PDL with significant bone loss.
- Conversion of bone marrow distant from the lesion to fibrous connective tissue.
- Widespread manifestation of inflammatory and immunopathologic tissue reaction.

Periodontitis is classified on the basis of extent and severity.

- Extent:
 - Localized = $\leq 30\%$ of sites involved
 - Generalized = $\geq 30\%$ of sites involved
- Severity can be characterized on the basis of the amount of clinical attachment loss (CAL):
 - Slight = 1 or 2 mm CAL
 - Moderate = 3 or 4 mm CAL
 - Severe = ≥ 5 mm CAL

Early Periodontitis

- Areas of localized erosion of the alveolar bone crest (blunting of the crest in the anterior regions)
- There is rounding of the junction between the crest and lamina dura in the posterior regions

Moderate Periodontitis

- The destruction of alveolar bone extends beyond early changes in the alveolar crest.

- May include buccal or lingual plate resorption, generalized horizontal erosion or localized vertical defects, and possible clinical evidence of tooth mobility.

Advanced Periodontitis

- The bone loss is so extensive that the remaining teeth show excessive mobility and drifting and are in jeopardy of being lost (see Fig. 8–6A).
- Characterized by extensive horizontal bone loss or extensive bony defects. X-ray shows significant bone loss between the two roots of a tooth (see Fig. 8–6B and 8–6C).
- The spongy bone has receded due to infection under tooth, reducing the bony support for the tooth.



FIGURE 8–6A. Advanced periodontitis.

Advanced Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)

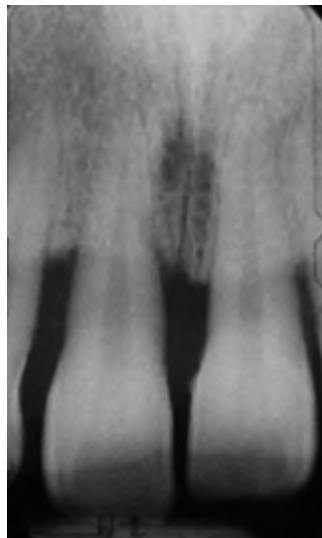


FIGURE 8–6B. Advanced periodontitis—50% bone loss—maxilla.

Advanced Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8–6 C. Advanced periodontitis—75% bone loss—mandible.

Advanced Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)

Aggressive Periodontitis

Occurs in two forms:

- **Generalized Form:**
 - *Prevotella intermedia* and *Eikenella corrodens* predominate
 - Occurs between ages of 12–25 and is characterized by rapid, severe periodontal destruction around most teeth
 - Characterized by episodic, rapid, and severe attachment loss
 - Weak serum response
- **Localized Form:**
 - Gram-negative anaerobes *Actinobacillus Actinomycetemcomitans* (AA) and *Capnocytophaga* species predominate although *P. intermedia* and *E. corrodens* may present to a lesser extent.
 - Strong serum response.
 - Occurs in otherwise healthy adolescents (ages 8–22).
 - Characterized by rapid and severe attachment loss confined to the incisors and first molars (see Fig. 8–7A to 8–7C).
 - Relative absence of local factors such as plaque to explain severe periodontal destruction.
 - Possibly etiologic factors include a genetic predisposition or a dysfunction of neutrophils (a chemotactic defect).

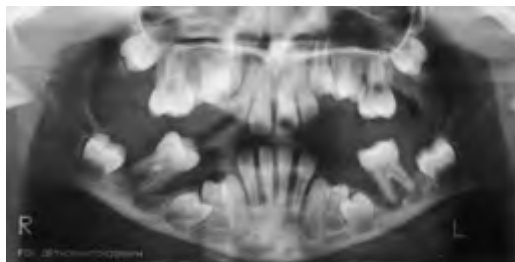


FIGURE 8-7A. Aggressive periodontitis panoramic X-ray, note affected incisors, and molars.

Aggressive Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-7B. Aggressive periodontitis; 11 years old with severe gingival inflammation.

Aggressive Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-7C. Aggressive periodontitis; 15 mm probing depth.

Aggressive Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)

Acute Necrotizing Ulcerative Gingivitis (ANUG)

- A condition which presents via pathognomic signs and symptoms.
- Characterized by interproximal necrosis and pseudomembrane formation on marginal tissues.
- Affects only the gingiva and is not associated with attachment loss.
- History of soreness, bleeding gums, and possible fetor otis (offensive odor), low grade fever, lymphadenopathy, and malaise.
- Affects mainly adults between the ages of 18–30 and is predisposed to patients with a history of smoking, gingivitis, poor oral hygiene, fatigue, stress, poor nutrition, and immuno-compromised patients.
- Treatment includes debridement, hydrogen peroxide rinses, and antibiotic therapy.

Acquired Immune Deficiency Syndrome (AIDS)

- Infection with human immunodeficiency virus (HIV).
- Primarily targets the CD4 helper lymphocytes leading to a severe state of immunosuppression.
- Macrophages are also affected.
- Patients are susceptible to a range of opportunistic bacterial, viral, protozoal, and fungal infections as well as neoplasms and neurological disturbances.
- Common oral manifestations are generalized candidiasis and hairy leukoplakia.

HIV infection is also associated with specific periodontal diseases.

HIV GINGIVITIS

- Characterized by a defined linear marginal gingival erythema (see Fig. 8–8).
- The gingivitis may become more generalized and is resistant to conventional therapy.



FIGURE 8–8. HIV gingivitis—note the linear gingival erythema.

HIV Gingivitis (Courtesy of Andrew Forrest, D.M.D., M.S.)

HIV PERIODONTITIS

- Rapidly progressive destruction of the gingiva with ulceration and cratering.
- Necrosis may be widespread, affecting soft and hard tissues, leading to sequestration of alveolar bone.
- The condition is extremely painful.
- Pathogenesis: Suppression of cellular host response mechanisms effectively increases the virulence of the periodontopathogens.
- Neutrophil function is not compromised as these cells are primed to a level of hyperactivity, possibly by the chronic exposure to bacteria.

Endotoxin

- Located on the cell wall of gram-negative bacteria consisting of a lipopolysaccharide base that has significant pathogenic potential.
- Capable of promoting bone resorption, inhibiting osteogenesis, chemotaxis of neutrophils, and other events associated with active periodontitis.
- Free endotoxin is found in dental plaque and inflamed gingiva.
- The involvement of bacterial endotoxins in gingival inflammation is supported by the fact that a reduction in inflammation by the removal plaque or a reduction of the inflammatory state via antibiotic treatment.

Attachment Loss

- Supportive structures are being destroyed.
- Pocketing can increase or decrease, depending on the amount of inflammation without attachment loss.
 - Extensive attachment loss and gingival recession may be accompanied by shallow pockets.

Periodontal Abscess

- Usually associated with acute pain that is constant, severe, and dull throbbing.
- Thermal changes do not seem to elicit or modify the discomfort.
- Onset of this pain is rapid and becomes progressively more intense with a potential increase in mobility of tooth.
- Abscess can be an exacerbation of chronic periodontal disease and may occur when the infection passes into the tissue through the pocket epithelium (see Fig. 8–9).

Three methods for treating periodontal abscesses:

1. Curettage via the sulcus.
2. Flap surgery.
3. Incision and drainage.

Plaque

- Soft deposit that accumulates on teeth consisting of a complex microbial community with greater than 10^{10} bacteria per milligram.
- Contains as well a small number of epithelial cells, leukocytes, and macrophages.



Attachment loss occurs in periodontitis, but not gingivitis.



Bitewing radiographs tend to be the most accurate in the determination of bone loss.



Antibiotics such as penicillin or clindamycin may be indicated pending patient manifestations and should be supported by culture and sensitivity where practical and indicated.



Patients with deep periodontal pockets and heavy deposits of plaque and calculus may develop an acute periodontal abscess after scaling. This occurs after scaling and root planning of deep sites the coronal tissue heals. The tissue contracts and reattaches and there may be infective material below. (Similar to the tightening of a purse-string).



FIGURE 8-9. Periodontal abscess associated with tooth #8.

Periodontal Abscess (Courtesy of Andrew Forrest, D.M.D., M.S.)

- Cells are contained within an extracellular matrix (dextran matrix), which is formed from bacterial products and saliva.
- Extracellular matrix contains protein, polysaccharide, and lipids.
- Dextrans are insoluble and sticky, which contributes to the ability of plaque to adhere to teeth.
- Inorganic components are also found in dental plaque: largely calcium and phosphorous (primarily derived from saliva).
- The inorganic content of plaque is greatly increased with the development of calculus.
- The process of calculus formation involves the calcification of dental plaque.
- The major factor in determining the different bacteria in supragingival and subgingival plaque is oxygen.
 - The redox potential of the gingival sulcus greatly influences the bacterial composition.

The attachment, growth, removal, and reattachment of bacteria to the tooth surface is a continuous and dynamic process. Several distinct processes can be recognized:

1. Absorption of salivary proteins and glycoproteins, together with some bacterial molecules, to the tooth surface to form a conditioning film called the **acquired pellicle**.
2. Long-range (>50 nm), nonspecific interaction of microbial cell surfaces with the acquired pellicle via van der Waals attractive forces.
3. Shorter range (10–20 nm) interactions, in which the interplay of van der Waals attraction forces and electrostatic repulsion produces a weak area of attraction that can result in reversible **adhesion** to the surface.
4. **Irreversible adhesion** can occur if specific intermolecular interactions take place between adhesins on the cell surface and receptors in the acquired pellicle.

5. Secondary or late colonizers attach to primary colonizers (**coaggregation**), also by specific intermolecular interactions.
6. Cell division of the attached cells to produce confluent growth, and a **biofilm** is formed.
 - In a biofilm such as dental plaque, microorganisms are in close proximity to one another and interact as a consequence. These gradients lead to the development of **vertical and horizontal stratifications** within the plaque biofilm.
 - Beneficial interactions include the concerted action of two or more species to metabolize host macromolecules, such as mucin (individual species are unable to catabolize such molecules), the development of food chains (e.g., lactate consumption by *Veillonella* spp), and coaggregation. Antagonistic interactions include the production of inhibitory substances such as bacteriocins, H_2O_2 , and organic acids.
 - Early colonizers of the tooth surface are mainly *Neisseria* spp. and *Streptococci*.
 - The growth and metabolism of these pioneer species change local environmental conditions (e.g., pH, coaggregation, substrate availability) enabling more organisms to colonize; for example, obligate anaerobes, which tend to be late colonizers in plaque, only able to grow once favorable gradients in O_2 or have developed in the biofilm.
 - Plaque develops naturally on teeth, and gives benefit to the host by providing colonization resistance. Once established at a site, the plaque flora remains relatively stable with time despite regular environmental challenges.
 - This stability (**microbial homeostasis**) is not due to any metabolic indifference by the resident microflora but is due to a dynamic balance being established among the resident members of this microbial community. On occasions, homeostasis breaks down and imbalances in the microflora can occur which predispose a site to disease.
 - In periodontal diseases, there is a shift in the composition of the plaque microflora to a more **proteolytic gram negative anaerobic community**, which can induce damage to tissues either indirectly via the *side effects* of an inflammatory host response or directly by the production of proteases, cytotoxins and other virulence factors.

Calculus

- Mineralized plaque formed by bathing plaque in a highly concentrated solution of calcium and phosphorous (i.e., saliva). Subgingival calculus is darker (due to pigments from blood breakdown), harder, and more dense than supragingival calculus.
- Supragingival calculus occurs above the free gingival margin:
 - Usually white or pale yellow in appearance and is easily removed by a professional cleaning
 - Occurs most frequently on the tongue side of the mandibular incisors (see Fig. 8–10) and cheek side of the maxillary molars
 - Usually occurs in these two locations due to the presence of salivary ducts which secrete saliva rich in minerals necessary for its formation
- Consists of inorganic components of calculus (70–90%); calcium and phosphates with small amounts of magnesium and carbonate (these are derived almost entirely from saliva).

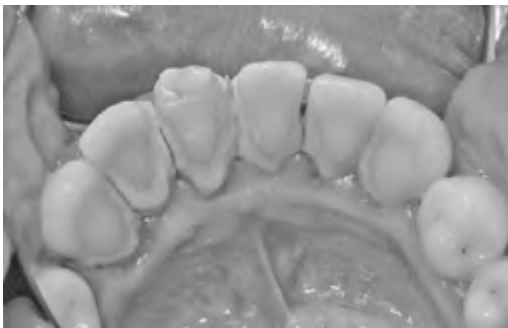


FIGURE 8-10. Calculus.

Calculus (Courtesy of Andrew Forrest, D.M.D., M.S.)

- At least two-thirds of the inorganic matter in calculus is crystalline, principally apatite; predominating is hydroxyapatite.
- Also contains octacalcium phosphate, tricalcium phosphate (whitlockite), and brushite.
- Organic components include microorganisms, desquamated epithelial cells, leukocytes, and mucin.
- Exerts its pathogenic potential as a contributing factor that fosters plaque formation and promotes its retention on teeth.
- Rough surface of calculus is usually covered with a layer of plaque biofilm.
- Calculus tends to “present” plaque via the biofilm to periodontal soft tissues.

Contributing Factors of Periodontal Disease

- Food impaction or retention due to overlapping, malposed, tilted, or drifted teeth.
- Open and loose contacts lead to food impaction and possible retention.
- Overhanging margins of restorations and improperly designed prosthesis can possibly initiate disease.
- Direct correlation between roughness of surface and retentive abilities of plaque.
- Soft or sticky diet of food debris can accumulate on teeth and along gingival margin.
- Violation of biological width can cause apical migration of junctional epithelium.
- Occlusal traumatism can exacerbate periodontal condition.



Access is the most important reason for performing periodontal surgery. Proper access allows for visualization of the roots so that the plaque and calculus may be removed more completely.

► PERIODONTAL SURGERY

Objectives of Periodontal Surgery

- Pocket reduction
- Promotion of gingival reattachment
- Treat and/or control the etiology
- Remove or eliminate the lesion
- Restore form and function

- Create an environment that can be maintained in health
- Balance the host's susceptibility/resistance against degree of insult

Reasons why periodontal surgery should ideally be performed after hygiene phase therapy has been completed:

- When pockets are due mainly to gingival hyperplasia, improvement in oral hygiene may result in resolution of inflammation and reduction in swelling, so surgery can be avoided.
- Reduction in inflammation means that bleeding at surgery is less of a problem.
- Where pockets are shallow (<5 mm), resolution following scaling and root planning may obviate the need for surgery.
- Flap surgery is contraindicated in patients who are unable to maintain adequate levels of plaque control.

Osseous Surgery

- Eliminate periodontal pockets by changing existing bony topography.
- Does not cure periodontal disease.
- Provides patient with opportunity and the access to maintain their periodontium and dentition with routine oral hygiene procedures (see Fig. 8-11A to 8-11D).



FIGURE 8-11A. Initial visit. (Also see color insert)

Osseous Surgery—Advanced Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-11B. Osseous surgery—flap reflected, debrided, scaled and root planned, roots conditioned, and bone recontoured. (Also see color insert)

Osseous Surgery—Advanced Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURES 8-11C and 8-11D. Osseous surgery reevaluation—6 week post-op visit; note accessibility for hygiene, tissue color, tone, consistency, and the return of gingival stippling. (Also see color insert)

Osseous Surgery—Advanced Periodontitis (Courtesy of Andrew Forrest, D.M.D., M.S.)

Other Treatment Modalities Considered

- Maintenance with periodic root planning
- Bone grafting (see Fig. 8-12A to 8-12D; Fig. 8-13A and 8-13B)
- Reattachment-fill procedures
- Hemisection or root amputation

The complications of periodontal surgery almost invariably results in a degree of gingival recession. This has a number of consequences:



FIGURE 8-12A. Preoperative.

Flap Surgery with Bone Grafting (Courtesy of Herbert J. Towle, III, D.D.S.)



FIGURE 8-12B. Flap elevated, teeth splinted, defects debrided, and visibility of the 75% attachment loss.

Flap Surgery with Bone Grafting (Courtesy of Herbert J. Towle, III, D.D.S.)



FIGURE 8-12C. Bone graft placed in defect.

Flap Surgery with Bone Grafting (Courtesy of Herbert J. Towle, III, D.D.S.)

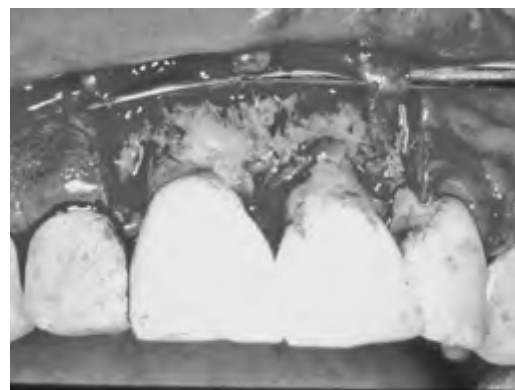


FIGURE 8-12D. Nine-month reevaluation and reentry.

Flap Surgery with Bone Grafting (Courtesy of Herbert J. Towle, III, D.D.S.)



FIGURE 8-13A. Preoperative radiograph on tooth #19.

Flap Surgery with Bone Graft—#19M (Courtesy of Andrew Forrest, D.M.D., M.S.)

- The increased exposure of root surfaces may lead to dentinal hypersensitivity.
- The increase tooth length may have compromised esthetics in the anterior region.
- The exposed root surface is more vulnerable to root caries.

Materials used for bone replacement grafts:

- Autograft: Bone taken from patient's own body, e.g., bone removed from the patient's hip.
- Allograft: Bone taken from another person—same species—e.g., bone harvested from cadavers and treated.
- Xenograft: Bone taken from another species—e.g., treated bovine (cow) bone.
- Alloplast: Synthetic bone—e.g., hydroxyapatite minerals or ceramics.



FIGURE 8-13B. Reevaluation of flap surgery with bone graft—defect fill 1 year radiograph.

Flap Surgery with Bone Graft—#19M (Courtesy of Andrew Forrest, D.M.D., M.S.)

Gingivoplasty

- Reshape the gingiva and papilla of a tooth for correction of deformities.
- Provide the gingiva with normal and functional form.
- Provide a physiological tissue contour.

Gingivectomy

- Procedure in which pocket depth is eliminated by resecting the tissue coronal to the pocket base.
- Amount of tissue removed depends on pocket depth and radiography.
- Creates a bevel/contour at the coronal margin of the surgical wound.
- Allows the patient to maintain adequate oral hygiene (see Fig. 8–14A and 8–14B).



FIGURE 8–14A. Initial visit.

Gingivectomy and Scaling (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8–14B. Gingivectomy reevaluation—1 month following gingivectomy, scaling, and oral hygiene instructions.

Gingivectomy and Scaling (Courtesy of Andrew Forrest, D.M.D., M.S.)

- Contraindications include infrabony pockets and a lack of keratinized tissue.
- Limitations include compromised esthetics with longer teeth, lack of access to bony defects, and having a broad, open wound postsurgically.

Modified Widman Flap

- Variation of the replaced flap.
- Full-thickness flap used for open flap debridement and regenerative procedures.
- Heals by repair usually via long junctional epithelium and connective tissue adhesion or attachment.
- Allows the clinician to gain access to the underlying bone and root surfaces.
- Reduces pocket depth by establishing a new attachment at a more coronal level.
- Preserves an adequate zone of attached gingiva.
- Provides an environment for healing by primary closure.

Apically Positioned Flap

- A full thickness mucoperiosteal flap is elevated and necessary treatment (scaling, root planing, osseous surgery, etc.) is completed.
- The gingiva is repositioned at the crest of bone.
- Usually requires vertical releasing incisions.
- The flap is repositioned to ensure no pocket remains following surgery.

Distal Wedge Procedures

- Removal of wedge reduces excess tissue and provides access for underlying bone.
- Performed in maxillary tuberosity region, mandibular retromolar triangle, and distal to the last tooth in arch.
- Many variations, but basic form is two incisions paralleling the outer gingival wall (which forms the wedge); the base being at the level of bone; the apex at the coronal gingival surface.

Guided Tissue Regeneration

- Creates a space via placement of a nonresorbable or resorbable barrier over a bony defect (see Fig. 8–15A and 8–15B).

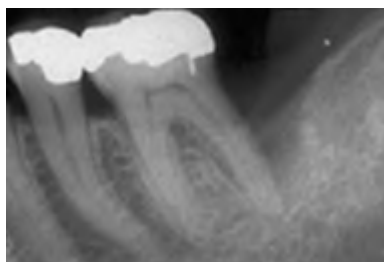


FIGURE 8–15A. Pre-op radiograph.

Guided Tissue Regeneration (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-15B. Guided tissue regeneration—1 year follow up.

Guided Tissue Regeneration (Courtesy of Andrew Forrest, D.M.D., M.S.)

- Prevents the ingrowth of long junctional epithelium and gingival connective tissue, allowing cells from the periodontal ligament and bone to repopulate the defect.
- Membranes include polytetrafluoroethylene (ePTFE) or resorbable membranes such as collagen, calcium sulfate, and polylactic acid.
- If a nonresorbable membrane is used, it must be retrieved 6–10 weeks after placement.
- Three-walled defects have the highest chance for success.

Osteoplasty

- Reshaping or recontouring bone that does not provide attachment for the periodontal fibers (nonsupportive bone)

Ostectomy

- Removal of osseous defects or infrabony pockets by elimination of the bony pocket walls.
- Bone removed may be supportive.
- This procedure allows the overlying gingival tissue to conform and be maintained.
- A major contraindication is removing crestal bone that may weaken the support of an adjacent tooth.

Root Amputation

- Separation of an individual root from the crown structure of the tooth. Burs are used to separate the crown and root prior to extraction by root tip forceps.
- The remaining apical area of the crown and furcation region is recontoured similar to the shape of a pontic so that the maximal access is provided for oral hygiene methods.
- The teeth that are most frequently involved in root amputation procedures are maxillary first and second molars (see Fig. 8-16A to 8-16D).



FIGURE 8-16A. Upper right quadrant: teeth #1 and #2 with hopeless prognosis.

Root Amputation (Courtesy of Mark Forrest, D.M.D.)



FIGURE 8-16B. Teeth #1 and #2 extracted; endodontic treatment completed prior to root amputation of distobuccal root of tooth #3.

Root Amputation (Courtesy of Mark Forrest, D.M.D.)



FIGURE 8-16C. 15 year reevaluation radiograph of tooth #3.

Root Amputation (Courtesy of Mark Forrest, D.M.D.)



FIGURE 8-16D. Distal of tooth #3 accessible for hygiene.

Root Amputation (Courtesy of Mark Forrest, D.M.D.)

Hemisection

- Using a bur, vertical splitting of the tooth through both crown and root.
- Ideally in mandibular teeth where the crown is divided through bifurcation region.
- One half of the tooth is extracted if one specific root has excessive loss in osseous support and the remaining half of the molar tooth now is treated as a premolar.
- Both root amputation and hemisection must be done in conjunction with endodontic therapy of the particular tooth.
- Root canal therapy is usually performed first.

Autogenous Free Gingival Graft

- Autogenous graft of gingiva that is placed on a viable connective tissue bed from which it receives nutrients.
- Donor tissue is harvested from an edentulous region or the palatal area.
- Used to prevent further recession.
- Can be used to effectively widen the attached gingiva (see Fig. 8-17A to 8-17D).
- The greatest amount of shrinkage occurs within the first 6 weeks.
- It is also useful for covering nonpathologic dehiscences and fenestrations.
- The primary reason for failure of a free gingival graft is disruption of the vascular supply before engraftment.
- The second most common reason is infection.

Connective Tissue Graft

- Autogenous subepithelial connective tissue graft is used to gain root coverage in cases of gingival recession (see Fig. 8-18A to 8-18C).

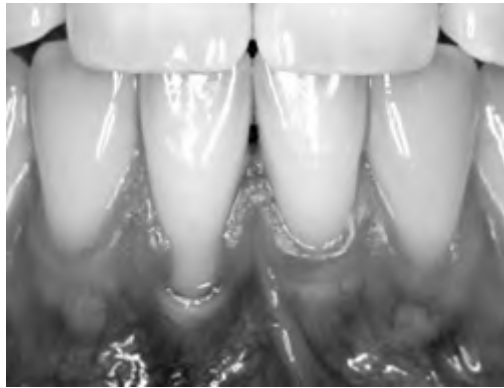


FIGURE 8-17A. Initial visit—3 mm recession on tooth #25—no keratinized or attached gingiva.

Free Gingival Graft—#25 (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-17B. Flap elevated; larger defect visible; root conditioned with 24% neutral pH EDTA.

Free Gingival Graft—#25 (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-17C. Gingival graft from palate fixated on recipient site.

Free Gingival Graft—#25 (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-17D. Six week reevaluation.

Free Gingival Graft—#25 (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-18A. Preoperative photo—6 mm recession.

Connective Tissue Graft (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-18B. Connective tissue graft—tunneling technique—graft inserted, 50% subgingivally and 50% exposed, fixated with cyanoacrylate.

Connective Tissue Graft (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-18C. Two months posttreatment reevaluation.

Connective Tissue Graft (Courtesy of Andrew Forrest, D.M.D., M.S.)

- The palate is also used to harvest the donor graft.
 - In this procedure the gingival epithelium is not used.
- This is a more predictable treatment modality for root coverage when compared to a free gingival graft.
- May be used to treat areas of sensitivity where gingival recession is the etiology.
- The causes of gingival recession are toothbrush or floss abrasion, high frenal attachments, factitious injury, bony dehiscence, thin alveolar bone, parafunctional habits, periodontal disease, natural consequence of aging, following periodontal treatment, prominent position of tooth in dental arch (i.e., buccal aspect of canine), and orthodontics (if the bands are improperly placed).

Flap

- A portion of marginal periodontal tissue that has been surgically separated coronally from its underlying support and blood supply and attached apically by a pedicle of supporting vascular connective tissue (see Fig. 8-19A and 8-19B).
- Full thickness mucoperiosteal flap
 - Includes surface mucosa (consisting of epithelium, basement membrane, and connective tissue lamina propria) and the contiguous periosteum of the underlying alveolar bone.
- Partial thickness flap
 - Includes only the mucosa, which is separated from the periosteum by sharp dissection.

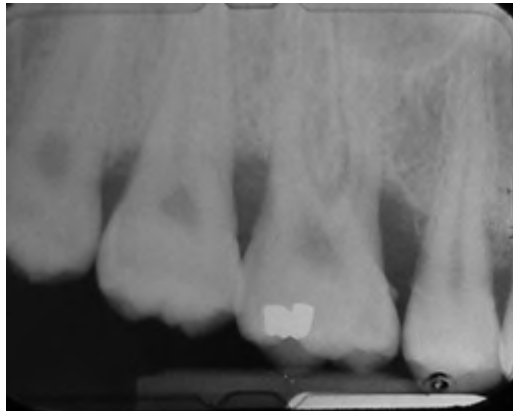


FIGURE 8-19A. Pre-op radiograph.

Flap Surgery (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-19B. Full thickness mucoperiosteal flap for access to defect; area débrided, scaled and root planed; more than 75% bone loss on tooth #3.

Flap Surgery (Courtesy of Andrew Forrest, D.M.D., M.S.)

FOUR BASIC RULES FOR FLAP DESIGN

1. Base of flap is wider than the free margin to provide adequate circulation.
2. Lines of incision should not be placed over any defect in the bone to prevent delayed healing.
3. Incisions that traverse a bony eminence should be avoided.
4. Corner of the flap should be rounded.

Crown Lengthening

- The procedure is indicated whenever clinical crown length is inadequate for the restoration (see Fig. 8-20A to 8-20F).
- The goal is to create a longer clinical crown for a tooth by removing both some of the gingiva and alveolar bone.



FIGURE 8-20A. Patient with temporary crown on tooth #5—initial visit.

Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-20B. Temporary crown removed.

Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-20C. Internal bevel incision.

Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S.)

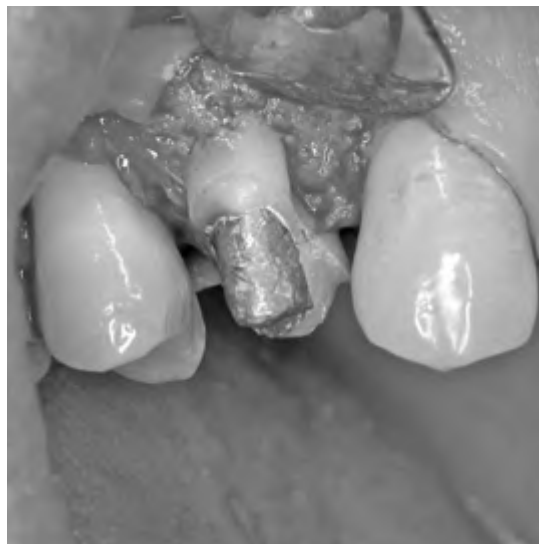


FIGURE 8-20D. Flap reflected—visibility of biologic width impingement on the mesial margin of tooth #5, approximately 1 mm from osseous crest.

Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-20E. Tooth crown lengthened; osseous structures recontoured.

Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S.)



FIGURE 8-20F. One month reevaluation—tooth ready for final prep, impression, and temporary crown.

Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S.)

- This surgical procedure frequently is the only option for salvaging a tooth crown that has been badly damaged by caries or fracture.
- The limiting factors are: crown to root ratio, esthetics, location of furcation, tooth–arch relationship.
- This measurement maintains a proper biologic width.
 - Biologic width—The connective tissue attachment (gingival fiber) and the junctional epithelium attachment to cementum are collectively known as the “biologic width.”
 - The quantified biologic width is 2.04 mm.
 - Connective tissue—1.04 mm (~1.00 mm)
 - Junctional epithelium—0.97 mm (~1.00 mm) (see Fig. 8–21A to 8–21D)



A general rule of thumb for a crown preparation is that you should have 3 mm between the margin of the preparation and the crest of bone to ensure adequate crown length. You will need 2 mm more for the prep to remain on sound tooth structure. The amount of tooth necessary required to expose for proper retention is 5 mm.



FIGURE 8–21A. Initial visit.

Esthetic/Cosmetic Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S. and Erica Elannan, D.D.S.)



FIGURE 8–21B. One month following crown lengthening.

Esthetic/Cosmetic Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S. and Erica Elannan, D.D.S.)



FIGURE 8-21C. Final prosthetics.

Esthetic/Cosmetic Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S. and Erica Elannan, D.D.S.)



FIGURE 8-21D. Two year reevaluation.

Esthetic/Cosmetic Crown Lengthening (Courtesy of Andrew Forrest, D.M.D., M.S. and Erica Elannan, D.D.S.)

Frenectomy

- A frenum is a band of muscle or fascia attaching the lips, cheeks, or tongue to alveolar ridges.
- Less than 1% require surgical intervention.
- Frenum problems occur most frequently in the following situations:
 - Localized gingival recession with frenum attachment on marginal gingival (high frenum attachment)
 - Midline diastema between two maxillary central incisors (low frenum attachment)
 - Ankyloglossia
 - Interference with denture fabrication
- The presence of a maxillary diastema does not prompt early frenectomy. Wait until the canines and laterals erupt before surgical intervention.
- Mandibular frenum problems should be treated when first noticed to prevent a mucogingival defect from developing.

Radiology

David Lee, DDS,
Timothy Leung, MHSc, DMD, MD

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Digital Radiology

Advances in computer and X-ray technology now permit the use of systems that employ sensors in place of X-ray films (with emulsion). The image is either directly or indirectly converted into a digital representation that is displayed on a computer screen.

DIGITAL IMAGE RECEPTORS

- The most common detector/sensor is the charged coupled device (CCD).
- Pure silicon divided into pixels.
- Electromagnetic energy from visible light or X-rays interacts with pixels to create an electric charge that can be stored.
- Stored charges are transmitted electronically and create an analog output signal and displayed via digital converter (analog to digital converter).

ADVANTAGES OF DIGITAL TECHNIQUE

- Immediate display of images.
- Enhancement of image (e.g., contrast, gray scale, brightness).
- Radiation dose reduction up to 60%.
- Major disadvantage: High initial cost of sensors. Decreased image resolution and contrast as compared to D speed films.

DIRECT IMAGING

- Radiographic image is acquired by a CCD or complementary metal oxide semiconductor (CMOS) detector that is sensitive to electromagnetic radiation.
- Performance is comparable to film radiography for detection of periodontal lesions and proximal caries in noncavitated teeth.

INDIRECT IMAGING

- Radiographic film is used as the image receiver (detector).
- Image is digitized from signals created by a video device or scanner that views the radiograph.

Sensors

There are different types of systems available in digital radiography. All systems share the same characteristic—the X-rays passing through the object are converted into a digital representation in a computer. There are two types that will be outlined although many systems employ hybrids of the two in varying degrees.

STORAGE PHOSPHOR IMAGING SYSTEMS

- Phosphor screens are exposed to ionizing radiation which excites BaFBR:EU+2 crystals in the screen storing the image.

- A computer-assisted laser then promotes the release of energy from the crystals in the form of blue light.
- The blue light is scanned and the image is reconstructed digitally.

ELECTRONIC SENSOR SYSTEMS

- X-rays are converted into light which is then read by an electronic sensor such as a CCD or CMOS.
- Other systems convert the electromagnetic radiation directly into electrical impulses.
- Digital image is created out of the electrical impulses.

Radiation Physics

DENTAL X-RAY TUBE

The dental X-ray tube is surrounded by a glass envelope that houses a vacuum. The glass prevents low-grade radiation from escaping. The vacuum insures the protection of the equipment from catastrophic failure. Production of X-rays generates enormous amounts of heat; the vacuum prevents the risk of combustion and ensures the proper environment for conduction of electrons. There are two separate energy sources, one that powers the energy potential between the cathode filament and the anode, and the other being the controls for the cathode filament. The latter essentially is the on and off switch of the X-ray unit. The cathode filament is heated which causes electrons to be emitted. These electrons are then accelerated by the electrical potential of the circuit. Between the two points is a tungsten target. When electrons strike the target, X-rays are produced.

HALF-VALUE LAYER

- Property of a material whereas the thickness (mm) reduces 50% of a monochromatic X-ray beam.
- Half-value layer of a beam of radiation from an X-ray unit is about 2 mm of aluminum (Al).

PRIMARY RADIATION

- Is the main beam produced from the X-ray tube.

SECONDARY RADIATION

- Produced by the collision of the main beam with matter which causes scatter.

Radiation Protection

FILTRATION

Filtration is a mechanism where the low quality, long wavelength X-rays are absorbed from the exiting beam. The process consists of aluminum disks in the X-ray tube that absorbs X-rays lower penetrating power. X-rays generated

from the target vary in wavelengths; filtering the long wavelengths reduces the overall dose received by the patient, the scale of contrast exposed on the film, and the density of radiation exposed to the film.

- **Two kinds of filtration:**
 - **Inherent filtration:** Absorption of the beam by glass, plastic cone, or aluminum filtration.
 - **Added filtration:** Additional aluminum disks are placed in cone to filter more lower grade radiation.

ULTRA-SPEED FILM (D, E SPEED FILM)

- The most effective form of protection for a patient is to use film that reduces exposure time and the amount of scatter radiation.

COLLIMATION

- Refers to the control of the size and shape of the emitted beam through the use of shaped cones in order to reduce the total area exposed to radiation.
- By law, the diameter of the beam must be no greater than 3 in.
- Distance from radiation exposure.

Radiobiological Concepts

Conventional Measurement	SI, Metric Equivalent
Rem	Sievert (Sv)
Rad	Gray (Gy)
Roentgen (R)	Coulomb

ABSORBED DOSE

- Measure of ionized radiation absorbed by an object
- Unit of measure is the **rad** (1 Gray = 100 rads)

EXPOSURE

- Measure of radiation quantity as it relates to the capacity of the ionizing radiation to ionize air. The measure of exposure is by the roentgen (R) or the Coulomb.

EQUIVALENT DOSE

- Measure of radiation quantity in tissue by a particular ionizing radiation.
- The measure is meant to be compared with other ionizing radiations in order to compare the biological effects.
- Measured by REM or Sievert (metric), 1 REM = 100 Sieverts.

EFFECTIVE DOSE

- Measure of the risk of ionizing radiation exposure to the body as compared to exposures to different parts of the body
- Measured in Sievert (metric)

RADIOSENSITIVE CELLS

- Radiosensitive cells undergo rapid mitosis or meiosis in the body and are therefore more susceptible to the damage caused by radiation.
- Reproductive cells.
- Lymphocytes.
- Bone marrow.

RADIORESISTANT CELLS

- Radioresistant cells typically do not undergo rapid mitosis or meiosis and are less susceptible to the damage caused by radiation.
- Muscle.
- Nerve.
- Mature bone.

OSTEORADIONECROSIS

- Radiation damages the bone producing necrosis of the exposed site.
- The mandible is the common site for osteonecrosis as the mandibular bone is less vascular than the maxilla.
- The period in between initial exposure and the first clinical sign of damage is called the “latency period.”

Radiographic Technique

FIVE RULES FOR ACCURATE IMAGE FORMATION WHEN TAKING X-RAYS

1. Use the correct size of focal spot; size of the focal spot is an inverse proportion to image qualities such as definition and sharpness.
2. Place the film at a distance close enough to the object to prevent distortion.
3. Use the longest target-film distance; this cuts down on divergent rays reaching the object. Most X-ray units use a 16 inch tube.
4. Central ray of the X-ray should be at a 90 degree angle to the film
5. The film and object should be made parallel to the object.

► IMAGE CHARACTERISTICS

Focal Spot

Source of X-rays caused by the collision between electrons generated from the filament onto the tungsten wire. Electrons generated from the filament travel down the potential from cathode to anode and collide between the two points at what is called the focal spot.

Target Film Distance

- The distance between the target (focal spot) to the film.
- Determines film density and exposure and ultimately film diagnostic quality.
- Decreasing this distance increases magnification of the object; it is preferable to use a 16 in. tube to decrease magnification and to increase the chance of parallel rays hitting the object.
- Increasing the target and film distance requires more time for exposure given that kilovoltage and milliamperage are held constant. This equation is given as

$$\frac{\text{Original Time}}{\text{New Time}} = \frac{\text{Original Distance}^2}{\text{New Distance}^2}$$

In real-life situations, this means a 16 in. tube requires more exposure time than a 8 in. tube, all factors being constant.

Magnification

- Caused by X-rays that are not parallel to the object or film.
- Can be caused by decreasing target-film distance and also increasing object to film distance.
- Orthopantograms, for example, panoramic images have a inherent 25% magnification in some areas of the focal trough.

Intensifying Screens

- Used in extraoral radiography for reducing the amount of radiation to the patient while retaining the same quality of picture.
- Most often, the emulsion film is sandwiched between two screens to further increase the quality and decrease the amount of exposure time and radiation used.

Radiopacity

- Object that inhibits or absorbs the passage of X-rays onto the film.
- Dense materials such as metals, bone, enamel, and dentin are radiopaque.
- Radiopaque structures appear “whiter” compared to radiolucent objects owing to less exposure of the film to X-ray particles.

Radiolucency

- Object that allows X-ray particles to pass through.
- Structures appear darker owing to more exposure of the film to X-ray particles.
- Radiolucent structures include soft connective tissue and the air spaces of the maxillary sinus.

Radiation Characteristics

Kilovoltage: Unit measure of electrical potential or the energy change between one point in a electric circuit to another. This translates to how fast the electrons move between one potential to another.

CHANGES IN KILOVOLTAGE

- Increasing kilovoltage reduces contrast on the film. This is a result of an increase in production of lesser penetrating X-rays as well as greater penetrating X-rays.
- Decreasing kilovoltage increases contrast on the film. X-rays produced are less varied in energy states resulting in either X-rays penetrating or not penetrating through the object and onto the film.
- **Inverse square law states** that the intensity of the film exposure decreases as a squared ratio as the distance between the object and the source of X-rays increases. Essentially, intensity increases or decreases exponentially as the source and object are moved while the distance between the object and the film remains the same.

$$\frac{\text{Original intensity}}{\text{New Intensity}} = \frac{\text{Original Distance}^2}{\text{New Distance}^2}$$

In a real-life situation, as you move the tube of the X-ray unit further from the object and film, the image becomes lighter due to decreasing intensity.

MILLIAMPERAGE

- The quantity or number of X-rays produced. This is determined by the temperature of the filament inside the X-ray unit.
- Density or the amount of exposure of a film is determined by how many particles of radiation are produced.
- Increasing the source to object distance, increasing the milliamperage, or increasing the exposure time increases density, which translates into increased exposure or “blackness” of the film.

Radiographic Errors

There are many ways to produce error on an X-ray film. The following are a list of tell-tale signs and their causes.

HERRINGBONE EFFECT OR THE TIRE TRACK PATTERN

- Characterized by a zigzagged pattern or the tire track pattern appearing on the X-ray film after development.
- Indicates the film was placed incorrectly with the exposed film facing away from the cone and the lead facing the cone.

FORESHORTENING AND ELONGATION

Foreshortening and elongation are terms that define errors in how much angulation in the vertical plane the head of the X-ray cone is compared to the object being examined. Foreshortening is characterized by the film image appearing *squashed* or shortened in the vertical. This is caused by too much vertical angulation of the cone in relation to the film. Elongation is the opposite; the image appears *stretched* in the vertical. This is caused by too little angulation of the cone to the object.

VERTICAL ANGULATION

- Refers to the direction of X-rays that will pass through an object in the vertical plane (superiorly, inferiorly).

HORIZONTAL ANGULATION

- Refers to the direction of X-rays that will pass through an object in the horizontal plane (anteriorly, posteriorly) such that the central ray is at 0 degrees to the object and 90 degrees to the anteroposterior plane of the object.

CENTRAL RAY

- Is the imaginary line that runs directly center of the X-ray cone.

POSITIVE ANGULATION

- When the tube of the X-ray unit is directed toward the ceiling, this is a positive angulation of the central ray.

NEGATIVE ANGULATION

- Or “minus” angulation refers to the cone being directed toward the floor.

Errors in Imaging

Errors in radiographs introduce images not normally seen in the normal architecture of the anatomy.

ARTIFACT

- An **artifact** is an image on the film introduced by the patient not removing objects in the field that would be radiopaque.

OVERBENT FILMS

- Appear as black semilunar radiopaquities or cracks in the film image.
- Is the result of bending the film as when a patient bites directly on the film.

LIGHT FILMS

- Caused by a number of errors such as incorrect distance from target to film distance, low milliamperage—these are problems of low density of exposure.

DARK FILMS

- Caused mainly by too much milliamperage, which results in too dense of an exposure of X-rays on film.

DOUBLE EXPOSURE

- Multiple images on the same film—the result of using the film more than one time.

FOGGED FILMS

- Cloudy films are the result of exposure from other sources of X-rays other than the main beam.

POOR CONTRAST

- Is the result of a kilovoltage setting that is too high resulting in increase of the scale of X-rays exposing the film.

BLURRED IMAGE

- Patient or cone movement during the exposure.

CLEAR FILMS

- Film was not exposed to X-rays.

Radiographic Techniques

PARALLELING TECHNIQUE

Technique of radiography where the central ray of the cone is directly perpendicular to the object and the film.

- Implies film must be parallel to the long axis of the tooth.
- Target to film distance must be optimum.

BISECTING TECHNIQUE

- Film is placed against the tooth with the central ray perpendicular to the bisecting line made from the angle of the film to the tooth.

Disadvantages of the bisecting technique are:

- Distortion caused by angulation is increased.
- Short cone used in bisecting technique increases divergent rays due to the shorter collimation produced by a shorter cone.

SUBMENTO-VERTEX TECHNIQUE

- Occlusal film is placed on the occlusal plane with the emulsion facing the chin. The central ray is then placed perpendicular to the film.
- Provides information on zygoma, zygomatic arches, and mandible. If done correctly, provides best diagnostic information on basilar skull fractures.
- The “jug-handle view” is a modification of this technique where the exposure is reduced to a third without any modifications in the technique. The zygomatic bones stand out in this view and the rest of the skull will be underexposed.

WATER'S VIEW OR PARANASAL SINUS VIEW (PNS)

- The patient is placed face toward the film with the chin angled superiorly against the film. The central ray of the X-ray is then directed perpendicular to the film.
- Offers diagnostic views of the maxillary, paranasal sinuses.

TOWNE'S VIEW

- Central ray is directed 30 degrees superior from the Frankfort plane with the film behind the patient's head.
- Offer views of the condylar head and the ramus of the mandible.
- Reverse Towne's view offers diagnostic views of condylar neck and ramus fractures.

PANORAMIC RADIOGRAPH

- The patient is placed in a room where the film and X-ray scan the patient from left to right. The condyles are viewed as well as the sinuses, and the third molars.
- Used for initial screening for pathology of the maxilla and mandible.

CERVICAL BURNOUT

- Radiolucent areas of interproximal teeth with ill-defined borders near the cervical aspect.
- Caused by normal architecture of the teeth in relation to decreased X-ray absorption in these areas; do not confuse with a carious process.

LATERAL HEAD RADIOGRAPH

- Film is placed to the side of patient head while the central ray is placed on opposite side of patient directed perpendicularly to the film.
- Used most often in cephalometric analysis and craniofacial growth; these are often used in orthodontics.

BITEWINGS

- Periapical film is placed perpendicular to occlusal plane with both mandibular and maxillary teeth in equal areas of exposure, and then the X-ray is directed perpendicular to the film.
- Vertical angulation should be at +8 or +10 to avoid overlap of the cusps.
- Best in diagnosis of caries and alveolar bone loss in the case of severe periodontitis.

SLOB RULE

Refers to a rule when using radiographs. For example, when viewing a foreign body at the roots of a tooth, the location of this foreign body is uncertain on a single periapical. The clinician must first assign a reference point, say the mesial root of tooth #19. Another periapical is taken except the central ray is off-set mesially by 60 degrees. If the foreign body is seen to move in the same

SLOB Rule
Same side Lingual
Opposite side Buccal

direction of the X-ray tube then the object is lingual to the reference point. If the object moves in the opposite direction then the object is buccal to the reference point.

Processing

DEVELOPING SOLUTION

DEVELOPING AGENT

- Main ingredient is hydroquinone which converts the exposed silver salts on the emulsion film into silver metallic ions while unexposed silver salts are left unaffected

ACCELERATOR

- Composed of an alkali salt such as sodium carbonate maintains the alkaline pH of the solution as well as provides ideal conditions for reactions to occur

RESTRAINER

- Composed of potassium bromide, this controls the actions of the developing agent such that unexposed silver salts are not removed from the emulsion film

ANTIOXIDANT PRESERVATIVE

- Present in fixing solution, serves to preserve the fixer from spontaneous oxidative processes
- Most often is sodium sulfite

FIXER SOLUTION

CLEARING AGENT

- Present in fixing solution, serves to rid the emulsion of underdeveloped silver salts
- Most often is sodium or ammonium thiosulfate

ANTIOXIDANT PRESERVATIVE

- Present in fixing solution, serves to preserve the fixer from spontaneous oxidative processes
- Most often is sodium sulfite

ACIDIFIER

- Present in fixing solution, serves to neutralize any alkaline developing agent carried over and also serves to buffer the solution and allowing certain chemical processes to proceed
- Most often is acetic acid

HARDENER

- Present in fixing solution, hardens the gelatin on the emulsion protecting it from physical damage and shortening the time it takes to dry the film postprocessing
- Most often is potassium alum

Film Development Problems	
Problem	Cause or Description
Weak Developing Solution	Over time images on film become lighter from overuse of developing solution.
Yellowish-Brown Film	Length of time in developer fluid determines whether a film is underdeveloped or overdeveloped. In this case, the film is not completely fixed at this point and needs more time in the developer to be fixed completely.
Fogged Film	Appears as general grayness on the film, indicating the presence of light in the darkroom or a faulty safety light.
Overdeveloped Film	Darkness in the film aside from incorrect calibration for the X-ray unit can be caused by incorrect timing of the wash, most likely too long in the developer, as well as the higher-than-normal wash temperature.
Underdeveloped Film	Aside from problems with the calibration of the X-ray unit, lightness of the film can be caused by a short wash in the developer, a temperature below the recommended level, or use of diluted solutions.
Torn Emulsion	Most often caused by contact between two films being processed, causing them to stick to each other. Separating the two causes a tear on the emulsion.
Stained Film	Appears as splotches on the film, caused by film being placed on a surface contaminated with chemicals.
Scratched Film	Film emulsion was abraded with sharp objects, most likely the fingernails of the person taking the film out of the sleeve.
Clear Films	Film was either not exposed or left in water for 24 hour period.
Air Bubbles	Appears as white spots on developed film, indicating that air bubbles were trapped on the surface of the film during processing.

► NORMAL RADIOGRAPHIC ANATOMY

See Figs. 9–1 through 9–9 for examples of normal radiographic anatomy.



FIGURE 9-1. Normal anatomy on occlusal films of the mandible.

1. Genial tubercles, 2. Mental ridge, 3. Lower border of the mandible

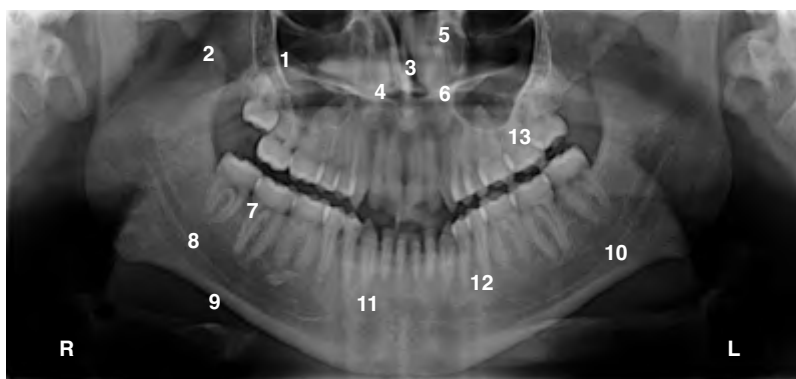


FIGURE 9-2. Normal anatomy on a panoramic X-ray.

1. Border of maxillary sinus, 2. Coronoid process, 3. Anterior nasal spine, 4. Nasopalatine fossa, 5. Lateral wall of nasopalatine, 6. Floor of nasal fossa, 7. Alveolar crest, 8. Mandibular canal, 9. Inferior border of mandible, 10. Submandibular gland fossa, 11. Mental foramen, 12. Extraction socket, 13. Floor of maxillary sinus

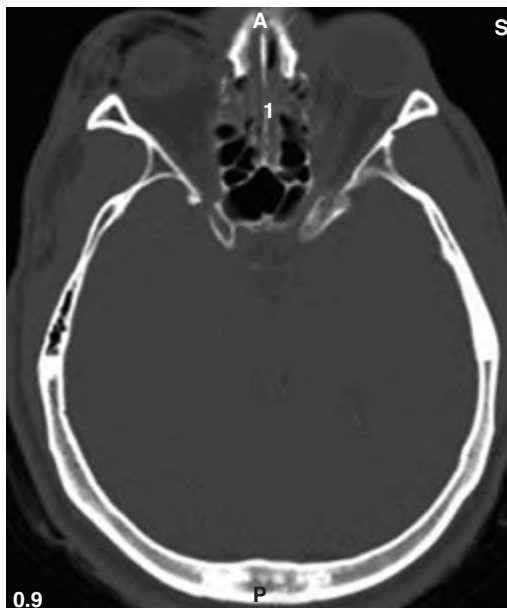


FIGURE 9-3. Axial head CT scan anatomy.

1. Vomerine bone



FIGURE 9-4. Axial head CT scan anatomy.

1. Lateral wall of maxillary sinus, 2. Medial wall of maxillary sinus, 3. Soft tissue tip of nose



FIGURE 9-5. Normal periapical film anatomy.

1. Pulp chamber, 2. Lamina dura, 3. Amalgam, 4. Cervical burnout, 5. Wall of maxillary sinus (Courtesy of Dr. Babak Zargari.)

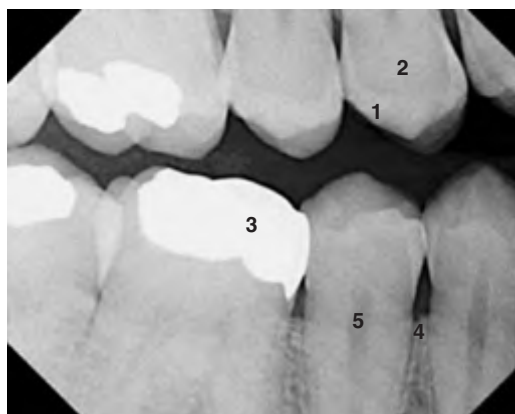


FIGURE 9-6. Normal bitewing film anatomy.

1. Enamel, 2. Dentin, 3. Amalgam, 4. Alveolar crest, sclerotic lamina dura, 5. Pulp chamber and canal (Courtesy of Dr. Babak Zargari.)



FIGURE 9-7. Normal mandibular periapical film anatomy.

1. Mandibular canal space, 2. Alveolar crest, 3. Pulp chamber, 4. Nutrient canal (Courtesy of Dr. Babak Zargari.)



FIGURE 9-8. Normal maxillary periapical film anatomy.

1. Mid palatine suture, 2. Nasal fossa, 3. Anterior nasal spine, 4. Incisive foramen (Courtesy of Dr. Babak Zargari.)

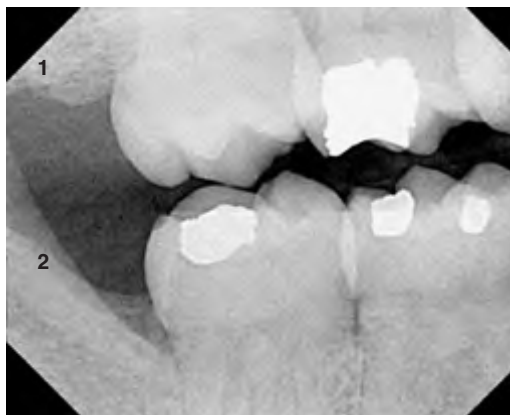


FIGURE 9-9. Normal posterior bitewing film.

1. Maxillary tuberosity, 2. Mandibular retromolar pad (Courtesy of Dr. Babak Zargari.)

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Agranulocytosis

A condition of severe neutrophil depletion, either because of inadequate production or because of increased destruction. Exposure to certain drugs (e.g., chemotherapeutics and antithyroid agents) causes most cases of agranulocytosis. Rare congenital syndromes such as congenital agranulocytosis and Kostmann syndrome can cause reductions in granulocyte colony-stimulating factor, thereby leading to agranulocytosis.

CLINICAL SIGNS AND SYMPTOMS

- Prodromal symptoms (malaise, sore throat, fever, chills).
- Swelling.
- Bone pain.
- Pneumonia.
- Punched-out ulcerations of the oral mucosa, tongue, and palate.
- Gingival infections resembling acute necrotizing ulcerative gingivitis.
- Histopathology of oral ulcerations typically shows lack of host inflammatory response.

TREATMENT

Removal of the offending drug as soon as possible is the first treatment. Oral hygiene along with chlorhexidine-containing mouth rinses should be implemented to decrease severity of oral ulcers. Antibiotics are indicated for any active infections. Granulocyte-macrophage-colony stimulating factor may be used for agranulocytosis related to cancer treatment, where the leukocyte count does not return to normal after cancer treatment.

Aplastic Anemia

The inability of the bone marrow to form adequate numbers of all types of blood cells. Hematopoietic cells do not undergo normal maturation. The cause of aplastic anemia is unknown. However, exposure to environmental agents (e.g., benzene), treatment with drugs like chloramphenicol, and exposure to viruses (e.g., hepatitis viruses, Epstein–Barr virus, HIV, and parvovirus) have been associated with the disease. In fact, aplastic anemia is the most serious blood disorder associated with drug toxicity.

CLINICAL SIGNS AND SYMPTOMS

- Pallor
- Fatigue
- Lightheadedness
- Malaise
- Dyspnea
- Headache
- Vertigo
- Spontaneous bleeding
- Bruising
- Oral mucosa petechiae
- Oral ulcerations
- Gingival infections

DIAGNOSIS

Pancytopenia with at least two of the following:

- <500 granulocytes/ μL
- $<20,000$ platelets/ μL
- $<10,000$ reticulocytes/ μL

TREATMENT

- Removal of causative agent
- Supportive therapies like antibiotics and blood transfusion for symptomatic treatment
- Androgenic steroids to stimulate bone marrow
- Immunomodulatory therapy (antilymphocyte globulin, corticosteroids, and cyclosporine)
- Bone marrow transplantation

PROGNOSIS

Unfortunately, despite cases of spontaneous recovery of bone marrow, aplastic anemia has a guarded prognosis at best. The mortality rate is $>80\%$ 1 year after diagnosis. Patients are typically at increased risk for acute leukemia.

Leukemia

This is a malignant neoplasm of the blood-forming tissues, characterized by an abnormal proliferation of leukocytes, which replaces tissue in the bone marrow with leukemic cells. The disease is further subdivided into categories depending on the hematopoietic stem cell derivation (myeloid vs. lymphocytic vs. monocytic) and the clinical course (acute vs. chronic).

CAUSES

Although the actual cause of leukemia is not completely determined, leukemias are associated with a combination of environmental, genetic, and chromosomal factors.

- Genetic disorders can include:
 - Town syndrome
 - Bloom syndrome
 - Klinefelter syndrome
 - Fanconi's anemia
 - Wiskott–Aldrich syndrome
- Chromosomal abnormalities can include:
 - Philadelphia chromosome $t(9;22)$ present in patients with CML
- Environmental agents include:
 - Benzene, benzene-like chemicals, aniline dyes
 - Ionizing radiation typically causes myelogenous forms of leukemia
- Viruses:
 - HTLV-1
- Epstein–Barr virus

CLINICAL SIGNS AND SYMPTOMS

When all types of leukemia are considered, the annual incidence rate is 13 cases per 100,000 population. Leukemic signs and symptoms are a presentation of the significant decrease in platelets, red and white blood cells, due to the malignant overproduction of leukemic cells. Symptoms include:

- Insidious onset (e.g., chronic leukemias) or abrupt onset (e.g., acute leukemias).
- Fever associated with infection may be initial presentation.
- Fatigue.
- Anemia.
- Dyspnea on mild exertion.
- Infiltration of other organs may cause organomegaly (e.g., liver, spleen) or lymphadenopathy.
- Thrombocytopenia, resulting in easy bruising and bleeding.
- Petechial hemorrhages of posterior hard palate and soft palate.
- Ulceration of oral mucosa, deep, punched out, gray–white necrotic base.
- Oral candidiasis common infection.
- Gingival hyperplasia due to infiltration of oral soft tissues by leukemic cells.
- Diagnosis is by peripheral blood smear and bone marrow biopsy, which will show poorly differentiated leukemic cells.

TREATMENT

Leukemia is treated with chemotherapy, radiation, and supportive therapy.

- **Induction chemotherapy:** High doses of chemotherapy to induce a state of remission
- **Maintenance chemotherapy:** Lower doses of chemotherapy administered over a longer period of time to maintain state of remission
- Radiation to the central nervous system since chemotherapy does not penetrate the blood–brain barrier
- Bone marrow transplant for those in remission
- Transfusions to manage bleeding problems
- Encourage optimal oral hygiene

PROGNOSIS

The outlook of these patients is dependent on age and type of leukemia.

- Acute myeloid leukemia (AML):
 - 10–30% 5 year survival.
- Acute lymphocytic leukemia (ALL):
 - Children have a 50–70% 5 year survival.
 - Adults have a 10–30% 5 year survival.
- Chronic myeloid leukemia (CML):
 - Eventually undergoes **blast transformation**. Death follows soon thereafter.
 - 60% 2 year survival after bone marrow transplantation.
- Chronic lymphocytic leukemia (CLL):
 - Considered incurable.
 - Those with limited disease may survive ≥ 10 years.
 - Those with more advanced disease may survive 2 years.

Polycythemia Vera

Also known as **Osler's disease** and **polycythemia rubra vera**, this is a hematologic condition where there is an increase in the blood volume as well as in the number of blood cells. The cells generally function normally. Patients are at a higher risk for leukemia.

CAUSES

- Primary polycythemia (abnormal multiplication of progenitor marrow stem cell)
- Secondary polycythemia (abnormal excess in erythrocytes due to other conditions such as hypoxia due to high altitudes, chronic pulmonary diseases, or erythropoietin secreting tumors)

CLINICAL SIGNS AND SYMPTOMS

The disease affects older adults and has no sex predilection, with approximately 4–16 cases per million population. Symptoms include:

- Headache
- Weakness
- Dizziness
- Dyspnea
- Epigastric pain
- Generalized pruritis
- A ruddy complexion
- Painful burning and erythema in the hands and feet
- Oral manifestations include:
 - Tongue and gingiva appear deep red
 - Gingival bleeding and swelling
 - Oral petechiae, ecchymoses, and hematomas
- Increased viscosity of the blood can lead to strokes, myocardial infarctions, hypertension, and splenomegaly

TREATMENT

- Phlebotomy
- Hydroxyurea, aspirin

Sickle Cell Anemia

This is a genetic hemoglobinopathy found predominantly in patients of African decent. A mutational substitution of a thymine molecule for an adenine in the DNA for hemoglobin results in the alteration of the amino acid glutamic acid into valine and a sickling of the red blood cell in the deoxygenated state. Patients inheriting one allele for sickle hemoglobin are **sickle cell trait carriers**. There are about 2.5 million people who carry the trait in the United States. Patients who inherit both alleles have **sickle cell disease**. In the United States about 1 out of 350 African Americans is born with sickle cell disease.

CLINICAL SIGNS AND SYMPTOMS

- Typical signs of anemia (e.g., dyspnea on exertion, fatigue, pallor).
- Muscle and joint pain.

- **Sickle cell crisis:** Patients present with acute ischemic pain typically in the long bones, lungs, and abdomen lasting for several days. The crisis may be precipitated by infection, hypothermia, hypoxia, or dehydration.
- ↑ susceptibility to *Streptococcus pneumoniae* infections.
- Impaired kidney function.
- “Hair-on-end” appearance on skull radiographs.
- Dental radiographs may show enlarged marrow spaces but are typically nonspecific.

TREATMENT

Patients are managed with supportive therapy, including fluids, rest, and analgesics. Prophylactic penicillin therapy and polyvalent pneumococcal vaccination may be indicated in children with sickle cell disease.

► NERVE AND MUSCLE DISORDERS

Bell's Palsy

Also known as seventh nerve paralysis and facial paralysis, this condition is an acute manifestation of unilateral paralysis of the muscles of facial expression.

CAUSES

Reactivation of herpes simplex virus within the geniculate ganglion is thought to cause most cases of Bell's palsy. Other triggers include:

- Exposure to cold
- Local and systemic infections
- Diabetes
- Influenza
- Tooth extraction

CLINICAL SIGNS AND SYMPTOMS

Facial signs of facial paralysis include ipsilateral:

- Inability to smile
- Inability to close the eye
- Inability to wink
- Inability to raise the eyebrow
- Drooping of the corner of the mouth
- Eye tears
- Slurring of speech
- Alteration of taste

TREATMENT

Symptoms typically resolve on their own after 1–2 months. Some studies have shown moderate improvement of symptoms with steroid and antiviral treatment. Ocular antibiotics can be used to prevent corneal ulceration.

Frey's Syndrome

Also known as **auriculotemporal syndrome**, this condition results from injury to the auriculotemporal nerve. The auriculotemporal nerve provides sympathetic sweat-stimulating fibers to the preauricular skin, in addition to parasympathetic fibers to the parotid gland.

CLINICAL SIGNS AND SYMPTOMS

When the nerve fibers to the parotid gland are damaged, in their attempt to reestablish innervation, they may be misdirected and reconnect with sympathetic fibers, leading to sweating, flushing, and warmth in the preauricular and temporal areas immediately following salivary or gustatory stimulation. **Minor's starch-iodine test** can be used to detect sweating in the preauricular area.

TREATMENT

Surgical severing of auriculotemporal or glossopharyngeal nerves, local atropine injections, or scopolamine creams can help decrease symptoms. Most cases are mild enough that long-term treatment is not required.

Glossopharyngeal Neuralgia

Similar to trigeminal neuralgia, this condition is characterized by unilateral, sharp, lancinating, and severe pain in the ninth cranial nerve distribution. The cause of glossopharyngeal neuralgia is unknown; however tumors and nerve compression along the glossopharyngeal nerve need to be ruled out.

CLINICAL SIGNS AND SYMPTOMS

Patients have episodic pain that comes about abruptly and lasts for short periods of time, typically less than 60 seconds. Pain typically presents in the ear canal, pharynx, nasopharynx, tonsils, posterior mandible, or posterior portion of the tongue. Triggers can include yawning, talking, chewing, and swallowing.

TREATMENT

Topical anesthetic to the tonsil and pharynx can help relieve pain, but pain relief is short term. Anticonvulsants (e.g., carbamazepine, phenytoin, or baclofen) and surgical resection of the ninth cranial nerve may be used for long-term relief.

Myasthenia Gravis

This is an autoimmune disease where circulating antibodies to acetylcholine receptors of the neuromuscular junction irreversibly attach to the receptors. Patients frequently also present with hyperplasia of the thymus or thymoma.

CLINICAL SIGNS AND SYMPTOMS

Progressive muscle weakness commonly seen in females and can occur at any age. Patients typically manifest global muscle weakness that progresses over the course of a day. In the head and neck this manifests as:

- Diplopia
- Ptosis
- Extraocular muscular paresis
- Weakening of muscles of mastication
- Dysphagia
- Dysarthria

TREATMENT

- Cholinesterase inhibitors (e.g., edrophonium, neostigmine)
- Thymectomy

Trigeminal Neuralgia

Also known as **tic douloureux** and **atypical facial pain**, this condition is characterized by severe pain in the head and neck region. Organic causes of pain such as inflammation, neuralgias, trauma, tumors, and sinusitis have typically been ruled out; however, the pain remains.

CAUSES

Typically this is idiopathic in nature.

CLINICAL SIGNS AND SYMPTOMS

This is the most frequently diagnosed neuralgia in the United States (four cases per 100,000 population). The pain is real and serious, sometimes leading to suicide attempts. The diagnosis of trigeminal neuralgia usually necessitates the fulfillment of certain criteria:

- Abrupt onset of pain, typically elicited by light touch (e.g., feather, strong breeze) over a specific trigger point
- Typical distribution is over one or more branches of the trigeminal nerve.
- Pain described as sudden, lancinating, and severe
- Each episode of pain is less than 60 seconds
- Carbamazepine helps relieve pain
- Remissions can last >6 months

TREATMENT

Quite variable treatment is used with little success.

- Anticonvulsant medications (e.g., phenytoin, carbamazepine)
- Local injection of alcohol or glycerin over nerve or trigeminal ganglion
- Local anesthesia of nerve or trigeminal ganglion
- Neurosurgery (e.g., severing sensory roots of trigeminal ganglion, decompression of trigeminal nerve)

► TOOTH ABNORMALITIES

Abrasion, Attrition, Erosion

Abrasion: Pathologic loss of tooth structure due to repetitive contact with of an external instrument like a toothbrush, pencil, or fingernail.

Attrition: Loss of tooth structure from tooth to tooth contact. To a certain degree, this is a physiological process, which increases with age. However, when the attrition begins to affect esthetic appearance and function, the process is pathologic. This can occur with poor occlusal contacts after dental restorations or from grinding habits.

Erosion: Loss of tooth structure from chemical processes (e.g., acid, gastric secretions, soft drinks).

CLINICAL SIGNS AND SYMPTOMS

- **Abrasion:** Toothbrush abrasion presents as horizontal notches on the buccal and cervical aspects of posterior teeth. Exposure of dentin and cementum is common, as is gingival recession. Biting of nails, pins, or pencils can result in V-shaped notches on the incisal edge of the incisors.
- **Attrition:** This typically affects surfaces in contact with the opposite dentition (e.g., occlusal surfaces, lingual surfaces of maxillary dentition, and labial surfaces of mandibular teeth). Wear facets can demonstrate the functional occlusal relationships.
- **Erosion:** Tooth loss does not correspond to functional wear patterns. Spoon-shaped depressions are seen in the cervical portions of maxillary anterior teeth. Posterior teeth show considerable losses on the occlusal surfaces, typically with a concave appearance since dentin erodes faster than enamel. In bulimics, the palatal surfaces of maxillary teeth typically present with exposed dentin.

TREATMENT

The main treatment is prevention through education on proper tooth brushing techniques and nutritional education. Tooth structure can be replaced by veneers, crowns, or composite resins. Pathologic occlusions and premature contacts should also be addressed.

Amelogenesis Imperfecta (AI)

One of the developmental alterations in the structure of teeth, this condition affects the development of enamel. The disease can be classified as **hypoplastic**, **hypomaturational**, **hypocalcified**, or both **hypomature and hypoplastic**. The different patterns of AI can be inherited in different ways, each with its own clinical presentation (Table 10–1).

CLINICAL SIGNS AND SYMPTOMS

- **Hypoplastic AI:**
 - Inadequate deposition of enamel matrix.
 - Enamel matrix present is normal.
 - **Generalized:** Small pits scattered across surfaces of teeth.
 - **Localized:** Large areas of hypoplastic enamel, typically on the buccal middle third of the tooth.
 - **Smooth:** Enamel exhibits a smooth surface, which is thin, hard, and glossy.
 - **Rough:** Enamel is thin, hard, and rough. The enamel is more dense than that of the smooth pattern.

TABLE 10-1. Inheritance Pattern and Special Clinical Features of Amelogenesis Imperfecta

TYPE OF AI	INHERITANCE PATTERN	CLINICAL FEATURES
Hypoplastic	Autosomal dominant	Pitted
	Autosomal dominant or autosomal recessive	Localized
	Autosomal dominant or X-linked dominant	Smooth
	Autosomal dominant	Rough
Hypomaturation	Autosomal recessive or X-linked recessive	Pigmented
	X-linked or autosomal dominant	Snow capped
Hypocalcified	Autosomal dominant or autosomal recessive	Coronal enamel loss
Hypomature and hypoplastic	Autosomal dominant	Taurodontism

- **Hypomaturation AI:**
 - Adequate deposition and mineralization of enamel matrix
 - Inadequate maturation of crystal structure
 - Soft enamel, with similar radiodensity to underlying dentin
 - **Pigmented:** Enamel has a mottled, brown appearance
 - **Snow capped:** Zone of opaque white enamel on the incisal/occlusal end of the tooth
- **Hypocalcified AI:**
 - No mineralization occurs
 - Enamel is soft and easily lost
 - Enamel and dentin appear radiographically indistinct
- **Hypomaturation and hypoplastic AI:**
 - In hypomaturation dominant pattern:
 - Enamel appears mottled and yellowish-brown
 - Pits on buccal surfaces
 - Enamel shows ↓ radiodensity
 - Taurodontism
 - In hypoplastic-dominant pattern:
 - Enamel is thin

TREATMENT

Depends on type and degree of AI.

- Veneers
- Full crown coverage
- Overdentures
- Full dentures

Ankylosis

Teeth continue to erupt even after emergence in to the oral cavity. When this eruption ceases, the tooth is considered to be ankylosed. Causes of ankylosis include trauma, abnormal pressure from the tongue, a smaller periodontal ligament (PDL) gap, disturbances in normal root resorption and hard tissue repair.

CLINICAL SIGNS AND SYMPTOMS

- Occlusal plane of the affected tooth is below the adjacent teeth.
- Radiographic absence of PDL.
- Adjacent teeth can migrate and incline toward the ankylosed tooth.
- Opposing teeth can migrate in vertical dimension, resulting in overeruption.

TREATMENT

- In the primary dentition, ankylosed molars should be extracted followed with space maintenance.
- In the permanent dentition, prosthodontic build-up.
- Luxation of ankylosed teeth can help reestablish a new PDL.

Dens Invaginatus

Also known as **dens in dente**, or “tooth within a tooth,” this is a deep invagination of the crown or root (see Fig. 10–1A). The invagination is lined with enamel. This most predominantly affects the maxillary lateral and central incisors, but can also affect the rest of the dentition.

CLINICAL SIGNS AND SYMPTOMS

There are typically few clinical symptoms associated with small invaginations. Larger openings may disrupt normal coronal formation. Invaginations that penetrate through the lateral root can cause periradicular inflammation. Invaginations that result in communication with the oral cavity put the tooth at increased risk for caries and pulpal necrosis. Radiographically, teeth have root enlargement, invaginations lined by radiopaque enamel, with variable extension into the tooth (see Fig. 10–1B).

TREATMENT

Complications related to dens invaginatus are rare. Invagination opening can be restored to prevent caries and pulpal necrosis. Large invaginations that disrupt normal coronal formation may need to be extracted. Invaginations associated with periradicular inflammation should be restored and have the surrounding lesion surgically removed.

Dentin Dysplasia

This condition describes a loss in the organization of the dentin in both deciduous and permanent teeth. There are two patterns of dentin dysplasia, type I and type II, with type I being the more common of the two. Dentin dysplasia is an autosomal dominant condition.

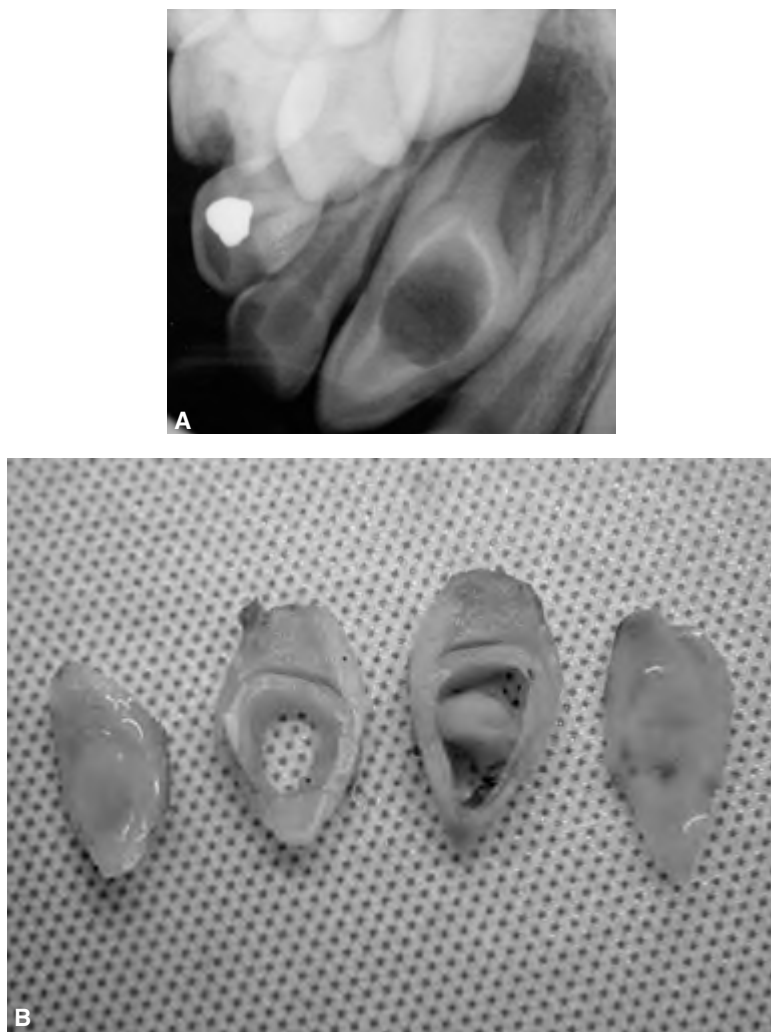


FIGURE 10-1. Dens in dente.

A. Malformed tooth-like structure with invaginated enamel and dentin layer extending to the root apex. Note associated peri-radicular radiolucency. B. Serial sections of the decalcified dens in dente (Courtesy of Drs. Robert Aslanian, Keith Murtagh, and John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

CLINICAL SIGNS AND SYMPTOMS

- **Dentin dysplasia type I:** Also referred to as “rootless teeth,” presents with normal coronal enamel and dentin, but the roots are shortened due to disorganized radicular dentin. The teeth are often mobile and there is premature exfoliation. The excess mobility can lead to increased risk of periodontitis and tooth loss. Radiographically, the pulp chambers of deciduous teeth are obliterated, while the pulp chambers in permanent teeth appear crescent-shaped.
- **Dentin dysplasia type II:** Resembles dentinogenesis imperfecta in that root length is normal, however, the crowns demonstrate a blue-brown translucence, are bulbous and have cervical constriction. Radiographically, deciduous teeth have obliterated pulp chambers, while permanent teeth have pulp chambers described as thistle tube and contain pulp stones.

TREATMENT

For both types of dentin dysplasia, meticulous oral hygiene is required to prevent periodontitis.

Dentinogenesis Imperfecta

A developmental alteration of teeth, this autosomal dominant condition affects the formation of dentin and may be seen in conjunction with **osteogenesis imperfecta**.

CLINICAL SIGNS AND SYMPTOMS

- Blue brown discoloration and translucence.
- Radiographic bulbous crowns with cervical constriction, and early obliteration of pulp chambers and canals.
- One variant has a shell tooth appearance with normal thickness enamel, thin dentin, and large pulp chambers.
- Root canals may develop microexposures resulting in periapical inflammation.
- Cervical fractures more common.

TREATMENT

- Full coverage of teeth with close to normal shaped crowns and roots
- Overlay dentures

Hypercementosis

This occurs when there is excess deposition of cementum on the normal radicular cementum. Hypercementosis has been associated with a host of different factors:

- Occlusal trauma
- Adjacent inflammation
- Paget's disease of bone
- Unopposed teeth
- Acromegaly
- Calcinosis
- Arthritis
- Goiter

CLINICAL SIGNS AND SYMPTOMS

The radiographic presentation includes:

- Thickening and/or blunting of roots
- Intact lamina dura
- PDL present
- Predominantly affects adults

TREATMENT

No treatment is required.

Internal and External Root Resorption

The loss of tooth structure from within the dental pulp (internal) is relatively rare, however, loss of tooth structure on the root adjacent to the periodontal ligament (external) is much more common.

CAUSES

Internal root resorption can be caused by injury to pulpal tissue related to trauma or to caries that penetrates to the pulp. External root resorption can be caused by:

- Dental trauma
- Orthodontic therapy
- ↑ occlusal forces
- Periradicular inflammation
- Periodontal treatment
- Cysts
- Tumors

CLINICAL SIGNS AND SYMPTOMS

Resorption of dentin and/or cementum can occur anywhere there is vital tissue in the adjacent areas.

- **Internal resorption** presents radiographically as a well-circumscribed radiolucency in the pulp chamber or root canal. Pink discoloration (**pink tooth of Mummery**) of the crown is observed when there is internal resorption in the pulp chamber.
- **External resorption** presents radiographically as a moth-eaten loss of root structure, typically in the apex or midroot portion of the tooth.

TREATMENT

- Remove tissue from sites of resorption
- Internal resorption: endodontic therapy
- External resorption:
 - Eliminate etiologic factors
 - Remove soft tissue adjacent to resorption site
 - Restoration of tooth structure

SUPERNUMERARY TEETH

Hyperdontia, or the development of an increased number of teeth, represents one of the developmental alterations in the number of teeth. Syndromes associated with hyperdontia include: cleidocranial dysplasia, Gardner, Sturge-Weber, Hallermann-Streiff, angio-osteohypertrophy, and Curtius.

CLINICAL SIGNS AND SYMPTOMS

Extra single teeth typically present in the permanent maxillary dentition, in the incisor region. Those in the maxillary incisor region are known as **mesiodens**. Multiple supernumerary teeth are frequently found in the premolar region of the mandible. Most supernumerary teeth fail to erupt, leading to delayed eruption in certain segments of the dentition. The extra teeth can

TABLE 10–2. Colors and Clinical Presentation of Extrinsic Tooth Staining

CAUSE OF STAIN	STAINING COLOR	CLINICAL PRESENTATION
Bacterial	Green Black-brown Orange	Frequently seen in children. Labial surfaces of maxillary anterior teeth.
Tobacco, tea, coffee	Brown	Lingual surfaces of mandibular anterior teeth.
Gingival hemorrhage	Green	Typically seen in conjunction with poor hygiene, hemorrhagic and edematous gingiva.
Restorative materials	Black-gray	Adjacent to large class II amalgam restorations or deep lingual metallic restorations.

also lead to crowding and malocclusion, predisposing these areas to gingival inflammation and periodontitis.

TREATMENT

Early removal of supernumerary teeth results in spontaneous eruption of adjacent teeth.

Tooth Staining

CLINICAL SIGNS AND SYMPTOMS

Tooth discoloration results from extrinsic sources (e.g., tobacco, coffee, red wine, amalgam, bacteria) (see Table 10–2) or intrinsic sources (e.g., amelogenesis/dentinogenesis imperfecta, fluorosis, medications) (see Table 10–3).

TABLE 10–3. Colors and Clinical Presentation of Intrinsic Tooth Staining

CAUSE OF STAIN	STAINING COLOR	CLINICAL PRESENTATION
Congenital erythropoietic porphyria	Reddish-brown	Red fluorescence on exposure to Wood's ultraviolet light.
Hyperbilirubinemia	Green Grayish-blue	Typically during the neonatal period of conditions like erythroblastosis fetalis and biliary atresia .
Dentinogenesis imperfecta	Gray Bluish-brown Translucent or opalescent	Radiographic appearance of shell teeth with normal thickness enamel and thin dentin.
Fluorosis	Opaque white Yellow-brown	Occurs during 2nd and 3rd years of life, with exposure to fluoride levels >1 part per million.
Medications	Bright yellow Dark brown Green Gray Black	Tetracycline exposure.

Clefting

Inappropriate development and merging of various tissue processes can lead to malunion of the lip, palate, or both.

- **Cleft lip** is caused by malunion of the **medial nasal process** with the **maxillary process**.
- **Cleft palate** results from failure of the **palatal shelves** to fuse.
- The majority of clefts are cleft lip with or without cleft palate, and isolated cleft palate.
- **Complete** cleft lip involves the entire lip and the underlying premaxilla, or alveolar arch.
- **Incomplete** cleft lip only involves the lip.
- Other less common cleft patterns include:
 - Lateral facial cleft
 - Oblique facial cleft
 - Median cleft of the upper lip
 - Median maxillary anterior alveolar cleft

CAUSES

- Syndromic
- Genetic
- Environmental factors

CLINICAL SIGNS AND SYMPTOMS

- Cleft lip with or without cleft palate is more common in males.
- Isolated cleft palate is more common in females.
- Most cleft lips are unilateral.
- Complete cleft lips include the nostril.
- Clefts involving the alveolar ridge usually occur in the area of the lateral incisor and canine.
- Cleft palate can involve the soft palate alone, or the soft and hard palate.

TREATMENT

- Primary lip closure in first few months of life
- Repair of palate usually between 12–18 months of age
- Interceptive orthodontics between 5–7 years of age
- Bone grafting for alveolar clefts at 9–11 years of age
- Comprehensive orthodontics and orthognathic surgery after 12 years

Rule of 10's for timing of cleft lip repair
 Infant should be:
 ≥10 weeks old,
 ≥10 g/dL hemoglobin, and
 ≥10 lb.

Cleidocranial Dysplasia

This autosomal dominant disease, also known as **cleidocranial dysostosis**, is a disease of the bones of the skull and clavicles.

CLINICAL SIGNS AND SYMPTOMS

- Short stature
- Large head with frontal bossing
- Ocular hypertelorism
- Hypoplastic midface
- Unusual mobility of the shoulders due to hypoplastic clavicles
- High arched palate
- Increased prevalence of cleft palate
- Prolonged retention of primary dentition (see Fig. 10–2)
- Unerupted teeth and supernumerary teeth present
- Narrow ascending ramus and pointed coronoid process

TREATMENT

There is no treatment for any of the bone anomalies. Dental treatment involves surgical exposure of unerupted teeth, orthodontic therapy, and possible orthognathic surgery and prostheses.

Cherubism

This is a rare inherited condition, with an autosomal dominant inheritance pattern, manifested by posterior mandibular swelling and plump cheeks.

CLINICAL SIGNS AND SYMPTOMS

- Painless, bilateral mandibular expansion, typically at the angle and rami.
- Maxillary involvement of the tuberosities.
- Developing teeth often displaced and fail to erupt.
- Radiographic bilateral multilocular radiolucencies in the mandible.
- Histopathologic manifestation similar to that of giant cell granulomas, well-vascularized fibrous tissue with multinucleated giant cells. Lesions in cherubism have a more loosely arranged appearance and perivascular eosinophilic cuffing.



FIGURE 10-2. Cleidocranial dysplasia.

Note numerous impacted supernumerary and permanent teeth. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

TREATMENT

- Some show remission and involution after puberty
- Some show remission with curettage of lesions, while some grow rapidly
- Definitive treatment has not been established

► METABOLIC JAW DISEASES

Osteopetrosis

This inherited condition results from a defect in osteoclastic bone remodeling and results in abnormally increased bone density. There are two major forms of the disease, infantile and adult type, with unique clinical presentations. The infantile type is inherited in an autosomal recessive pattern, while the adult type is autosomal dominant.

CLINICAL SIGNS AND SYMPTOMS

- **Infantile osteopetrosis**
 - Facial deformities:
 - Broad face
 - Hypertelorism
 - Frontal bossing
 - Flattened nose
 - Delayed tooth eruption
 - Increased susceptibility to infections due to marrow failure and subsequent granulocytopenia
 - Increased susceptibility to osteomyelitis after extractions
 - Narrowing of skull foramina can lead to:
 - Blindness
 - Facial paralysis
 - Deafness
 - Pathologic fractures are common
 - Compensatory extramedullary formation of blood cells leading to:
 - Anemia
 - Hepatosplenomegaly
 - Radiographic loss of cortical and cancellous bone distinction
 - Poor prognosis, usually resulting in death from anemia or infection before age 20
- **Adult osteopetrosis**
 - Milder form
 - ↓ frequency of anemia
 - Blindness, deafness, and facial paralysis due to narrowing of skull foramina
 - Fractures
 - Osteomyelitis and fracture of mandible can still occur

TREATMENT

There is no definitive therapy for osteopetrosis, aside from supportive measures, such as antibiotics (for infections) and blood transfusions (for anemia).

Osteogenesis Imperfecta

This inherited connective tissue disease results from impaired collagen synthesis.

CLINICAL SIGNS AND SYMPTOMS

Type	Inheritance Pattern	Clinical Manifestation
I	Autosomal dominant	■ Mild to moderate bone fragility ■ Hearing deficits ■ Blue sclera ■ Opalescent dentin (dentinogenesis imperfecta)
II	Autosomal recessive Autosomal dominant	■ Extreme bone fragility ■ Frequent fractures ■ 90% are stillborn ■ Blue sclerae ■ Opalescent dentin (dentinogenesis imperfecta)
III	Autosomal recessive Autosomal dominant	■ Severe bone fragility ■ Blue sclerae in infants ■ ↑ mortality in older children ■ Opalescent dentin (dentinogenesis imperfecta)
IV	Autosomal dominant	■ Mild to moderate bone fragility ■ Pale blue sclerae in early childhood ■ Fractures at birth ■ Opalescent teeth

TREATMENT

There is no curative treatment for osteogenesis imperfecta. Teeth are typically weakened due to coexistence of dentinogenesis imperfecta, and require extensive restorative work.

► ENDOCRINE DISORDERS

Hypoparathyroidism

A decrease in parathyroid hormone (PTH) level can be caused congenital disorders, iatrogenic causes (e.g., drugs, removal of the parathyroid glands), infiltration of the parathyroid glands, suppression of parathyroid function, HIV/AIDS, or idiopathic mechanisms. The level of calcium in the body is controlled by PTH. The hormone acts on osteoclasts of the bone to help resorb bone and increase levels of calcium in the blood.

CLINICAL SIGNS AND SYMPTOMS

- Hypocalcemia
- Chvostek's sign:
 - Twitching of the upper lip when the facial nerve is tapped
- Pitted enamel hypoplasia
- Failure of tooth eruption

TREATMENT

Oral vitamin D precursor and supplementary calcium are typically prescribed to normalize calcium levels.

Hyperparathyroidism

Increases in PTH can be caused by tumors of the parathyroid glands, parathyroid hyperplasia, or in response to chronically low levels of calcium in the body (e.g., chronic renal failure, rickets, and malabsorption syndromes). **Primary hyperparathyroidism** is usually caused by a parathyroid adenoma or hyperplasia. In rare cases, primary hyperparathyroidism is caused by a parathyroid carcinoma. **Secondary hyperparathyroidism** is caused by chronic low levels of serum calcium (e.g., chronic renal disease → vitamin D deficiency → poor gastrointestinal [GI] absorption of calcium).

CLINICAL SIGNS AND SYMPTOMS

- Stones: ↑ tendency of kidney stones, calcification in soft tissues.
- Bones: Resorption of bones of index and middle fingers, loss of lamina dura around roots, "ground glass" appearance of trabeculae.
- Groans: Duodenal ulcers.
- Moans: Mental status changes such as confusion, lethargy, confusion, or dementia.
- **Brown tumor of hyperparathyroidism** occurs with persistent hyperparathyroidism.
 - Osseous lesion
 - Radiographically appears as well-circumscribed unilocular or multilocular radiolucency
 - Commonly seen in the mandible, clavicle, ribs, and pelvis
 - Histologically similar to central giant cell granuloma

TREATMENT

Primary hyperparathyroidism is treated surgically. Secondary hyperparathyroidism from chronic renal failure is treated with vitamin D or renal transplantation.

Hyperthyroidism

Excess production of thyroid hormone is caused by thyroid tumors, hyperplastic thyroid tissue, autoimmune stimulation of thyroid cells (**Graves' disease**), and pituitary adenomas.

Clinical signs and symptoms of hyperparathyroidism
Renal stones
Painful bones
Abdominal groans
Psychic moans

CLINICAL SIGNS AND SYMPTOMS:

- More frequently seen in females.
- Thyroid enlargement.
- Increased metabolic rate, leading to:
 - Weight loss
 - Tachycardia
 - Warm skin
 - Heart palpitations
 - Tremors
 - Heat intolerance
- Protrusion of the eyes.
- Characteristic stare with lid lag and lid retraction.
- Elevated thyroid stimulating hormone levels and thyroxine (T_4).
- **Thyroid storm:** Uncontrolled hyperthyroidism can lead to delirium, elevated temperatures, tachycardia, and death.

TREATMENT

- Radioactive iodine
- Propylthiouracil
- Methimazole
- Surgery (thyroidectomy)

Hypothyroidism

Also known as **cretinism** during infancy, and **myxedema** in the adult, this is a condition characterized by low levels of thyroid hormone. Adult hypothyroidism is often caused by **Hashimoto's thyroiditis**, an autoimmune destruction of the thyroid gland. **Primary hypothyroidism** occurs when there is inadequate production of thyroid hormone because there is an abnormality in the thyroid gland itself. **Secondary hypothyroidism** occurs as a result of inadequate production of thyroid stimulating hormone (TSH) by the pituitary gland. Often seen after radiotherapy for brain tumors.

CLINICAL SIGNS AND SYMPTOMS

- Lethargy
- Dry and coarse skin
- Swelling in the face and extremities
- Bradycardia
- ↓ body temperature
- Skin feels cool to touch
- Thickened lips
- Enlarged tongue
- Failure of tooth eruption, impaired tooth formation

TREATMENT

- Thyroid replacement therapy (levothyroxine).
- Timely replacement is important in cretinism to prevent permanent damage to the central nervous system.

Acromegaly

This is a condition caused by the excessive production of growth hormone due to a functional pituitary adenoma. This results in continued growth of the skeleton despite closure of the epiphyseal plates.

CLINICAL SIGNS AND SYMPTOMS

- Hypertension, heart disease, peripheral neuropathy, arthritis, and excess sweating
- Headaches and visual disturbances from space occupying tumor in the head
- Growth in hands, feet, skull, and jaws (mandibular prognathism)
- Patients complain of gloves or hats not fitting anymore
- Coarse facial appearance
- Hypertrophy of soft palate
- Macroglossia
- Anterior open bite
- ↓ interdental spacing

TREATMENT

- Pituitary tumor removal.
- Radiation therapy in some cases.
- Octreotide (a somatostatin analogue) is used for patients in whom surgery is contraindicated.

Paget's Disease of Bone

This disease causes abnormal resorption and deposition of bone. Bones become weakened and distorted as a result of this dysfunction in bone remodeling.



Paget's disease—cotton wool appearance.

CLINICAL SIGNS AND SYMPTOMS

- Most cases are polyostotic (more than one bone affected), but can also be monostotic
- Bone pain
- Lumbar vertebrae, skull, femur, and pelvis enlargement:
 - Bowing in the legs, simian stance
 - Increase in head circumference
- Maxillary involvement > mandibular involvement
 - Lion-like facial deformity
 - ↑ interdental spacing in the maxilla
 - Dentures may no longer fit
- Radiographic decrease in bone density and trabeculation
 - Cotton wool appearance
 - Hypercementosis of teeth
 - There is increased vascularity in bone and is warm to touch
 - ↑ alkaline phosphatase levels with normal blood calcium and phosphorus
 - Osteosarcoma noted in approximately 1% of cases

TREATMENT

- Analgesics for bone pain.
- Periodic reevaluation of dentures.
- Calcitonin and bisphosphonates are PTH antagonists, and are used to reduce bone turnover.

Scleroderma

Also known as **systemic sclerosis**, this relatively rare condition is characterized by the excessive deposition of collagen in various tissues of the body (e.g., skin, organs). Although not completely understood, it is thought that the condition has an immunologically mediated pathogenesis.

Clinical Signs and Symptoms

- More frequently found in women, with adult onset.
- Diffuse form can present as pulmonary fibrosis, cor pulmonale, acute renal failure, and malignant hypertension.
- Involvement of facial skin presents a tight “mask-like” facies.
- Microstomia due to collagen deposition in perioral tissues.
- Loss of attached gingival mucosa; gingival recession.
- Dysphagia from collagen deposition in the lingual and esophageal submucosa.
- Diffuse widening of periodontal ligament space.
- Resorption of posterior ramus of the mandible, coronoid process, and condyle due to pressure from abnormal collagen production in adjacent areas.
- Inelasticity of mouth may make dental prostheses difficult to insert and remove.
- Oral hygiene may also be difficult to maintain for same reason.
- Diagnosis:
 - Rheumatoid factor (RF) and antinuclear antibodies (ANA) may be positive
 - Anticentromere antibodies associated with CREST syndrome (limited form)
 - Anti-Scl 70 (antitopoisomerase I) antibodies seen in more systemic cases

TREATMENT

- Steroids for acute flares.
- D-penicillamine used to inhibit collagen production.
- Esophageal dilatation can be used for symptomatic dysmotility.
- Calcium channel blockers for Raynaud’s.

CREST syndrome

Calcinosis cutis
Raynaud’s phenomenon
Esophageal dysmotility
Sclerodactyly
Telangiectasias

CREST Syndrome

An uncommon condition that represents a mild variant of scleroderma. The term represents an acronym for the following manifestations: calcinosis cutis, Raynaud’s phenomenon (usually appears first), esophageal dysmotility, sclerodactyly, and telangiectasias.



FIGURE 10-3. CREST syndrome, telangiectasias on the dorsal tongue. (Also see color insert)

The patient's hands showed sclerodactyly and Raynaud's phenomenon. (Courtesy of Drs. Stanley Kerpel and John Kacher.)

CLINICAL SIGNS AND SYMPTOMS

- Calcinosis cutis consists of multiple movable, nontender subcutaneous nodules.
- Raynaud's phenomenon occurs when hands and feet become white in color when exposed to cold temperatures as a result of inappropriately severe vasospasm. The affected extremity can become blue as a result of venous stasis. Upon warming, the extremity becomes red and may be accompanied by a throbbing pain.
- Esophageal dysfunction occurs as a result of abnormal collagen deposition in the esophagus submucosa.
- Sclerodactyly appears as claw-like fingers, due to abnormal collagen deposition in the fingers.
- Telangiectasias can manifest as red facial macules on the face and vermillion borders of the lips (see Fig. 10-3).

TREATMENT

Similar treatment as that for scleroderma, but may be less aggressive due to its diminished severity in comparison to scleroderma.

► INFLAMMATORY JAW LESIONS

Periapical Abscess

This process involves an accumulation of inflammatory cells at the apex, or adjacent to, a nonvital tooth. Periapical abscesses can be categorized symptomatic or asymptomatic depending on the presence or absence of pain.

CLINICAL SIGNS AND SYMPTOMS

- Accumulation of purulent material in alveolus adjacent to tooth.
- Sensitivity to percussion and palpation.
- Swelling of adjacent tissues.



Abscesses associated with maxillary lateral incisors, palatal roots of maxillary molars drain toward the palate, and mandibular second and third molars drain through the lingual cortical plate.

- Ø response to cold test or electric pulp testing.
- Headache, malaise, fever, chills.
- When abscesses perforate, they typically perforate through the buccal plate.

TREATMENT

- Extraction or root canal therapy
- Analgesics and antibiotics for severe cases

Periapical Granuloma

Chronically inflamed granulation tissue at the apex of nonvital teeth, secondary to the presence of bacteria in the root canal. The reaction eliminates noxious substances that exit the canals. With time, the host reaction weakens and becomes less effective with microbial invasion. These lesions may arise following quiescence of a periapical abscess and may also transform into periapical cysts.

CLINICAL SIGNS AND SYMPTOMS

Periapical granulomas are usually asymptomatic. Pain and sensitivity can develop. Radiographically, the lesion presents as a well-circumscribed radiolucency associated with root apices. Root resorption is sometimes observed as well.

TREATMENT

If the tooth can be maintained, root canal therapy can be initiated. Nonrestorable teeth with periapical granulomas need to be extracted. Unless the teeth are symptomatic after root canal therapy, patient's can be evaluated at 1 and 2 year intervals to rule out any enlargement of the lesion. Lesions that do not resolve following endodontic therapy should be biopsied and have apicoectomy performed.

Osteomyelitis

This is an inflammatory process involving the medullary space of bone, typically originating from a bacterial source. Cases of osteomyelitis are often the result of chronic odontogenic infections. Acute necrotizing ulcerative gingivitis has also been linked with osteomyelitis in developing countries. Other risk factors include immunocompromised state, decreased bone vascularity, and chronic systemic diseases. Radiation, osteopetrosis, and late stage Paget's disease can diminish vascularity to the bone, thus leading to increased risk of osteomyelitis.

CLINICAL SIGNS AND SYMPTOMS

- Predominantly found in males
- Mandible is frequent site
- Acute osteomyelitis:
 - Pain
 - Fever
 - Leukocytosis

- Lymphadenopathy
- Soft tissue swelling
- Increased sensitivity to palpation of affected bone
- Radiographs may show ill-defined radiolucency
- Chronic osteomyelitis:
 - Pain
 - Swelling
 - Sinus formation
 - Sequestration formation.
 - Purulence
 - Pathologic fractures
 - Radiographs may show patchy, ragged, ill-defined radiolucencies, with surrounding radiodensity

TREATMENT

- Acute osteomyelitis:
 - Antibiotics for oral flora (e.g., penicillin, clindamycin, cephalexin, cefotaxime, and gentamicin).
 - Drainage.
- Chronic osteomyelitis:
 - Surgical intervention is mandatory.
 - Antibiotics for oral flora.
 - Removal of infected and necrotic bone.
 - Replacement of large amounts of bone is accomplished by transplantation of cancellous bone chips or autologous graft.
 - Immobilization of the jaws may be required for weakened bones.
 - Hyperbaric chamber for patients who are resistant to standard therapy.

Condensing Osteitis

Also known as focal **sclerosing osteomyelitis**, this is a condition that involves bony sclerosis around the roots of teeth with associated pulpal infection (e.g., pulpitis or pulpal necrosis). Commonly seen in children and young adults.

CLINICAL SIGNS AND SYMPTOMS

Radiographic presentation of this process shows localized radiodensity around teeth roots as well as a thickening of the associated periodontal ligament. The teeth most often affected are the mandibular premolars and molars.

TREATMENT

Treat the odontogenic infection either via tooth extraction or endodontic therapy. A persisting lesion may require endodontic retreatment.

► CONNECTIVE TISSUE LESIONS

Pyogenic Granuloma

This vascular proliferation is more commonly seen in females. When it occurs in pregnant women, it is referred to as a **pregnancy tumor**.



Peripheral giant cell granulomas and peripheral ossifying fibromas present exclusively on the gingiva.

CLINICAL SIGNS AND SYMPTOMS

Frequently found on the gingiva, this is an overgrowth of markedly well-vascularized fibrous connective tissue secondary to irritation or trauma (see Fig. 10–4). The lesion is in actuality a vascular proliferation rather than a granuloma.

TREATMENT

Treatment is conservative surgical excision. Dental cleanings are effective for removing calculus and other sources of irritation. In a pregnant female, treatment may be delayed until after delivery as lesions may regress. Recurrent bleeding may necessitate excision and cauterization.

Peripheral Giant Cell Granuloma

Also known as giant cell epulis, this is a common proliferative growth in the oral cavity with a similar clinical appearance to the pyogenic granuloma. Local irritation or trauma is usually the cause of a peripheral giant cell granuloma. It is histologically identical to central giant cell granuloma, but is found in the soft tissue rather than the bony tissue. Peripheral giant cell granulomas appear exclusively on the gingiva or edentulous alveolar ridge. Can appear exophytic, sessile, red, blue, or purple. Similar to the pyogenic granuloma, there is a female predilection. Treatment usually involves careful surgical removal of the lesion down to the bone. Adjacent teeth are cleaned to remove possible sources of irritation. Recurrence rates are about 10%.

CLINICAL SIGNS AND SYMPTOMS

- Presents on gingiva or alveolar ridge
- Reddish blue nodular mass
- More frequently found in females (60%)
- May see “cup-like” resorption of the underlying alveolar bone
- Typically, an ulcerated mucosal surface is observed

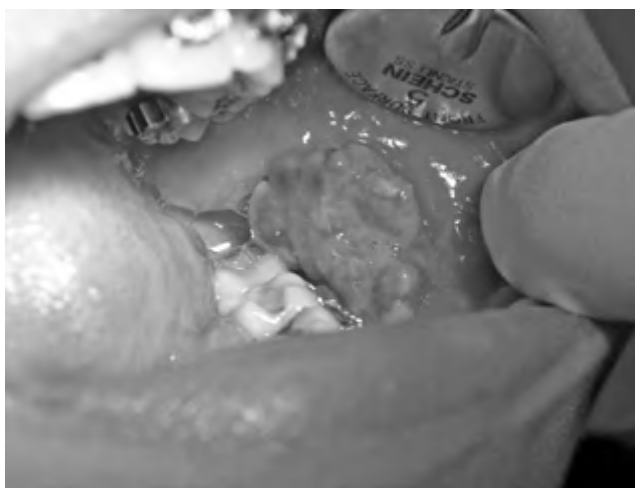


FIGURE 10–4. Pyogenic Granuloma. (Also see color insert)

Large lobulated, pedunculated lesion of the left buccal mucosa in a pregnant woman in her third trimester. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

TREATMENT

- Local surgical excision down to bone
- Remove local sources of irritation (e.g., calculus in adjacent teeth)

Peripheral Ossifying Fibroma

This lesion is believed to be a reactive fibrous proliferation and does not represent the peripheral variant of central ossifying fibroma.

CLINICAL SIGNS AND SYMPTOMS

- Presents exclusively on the gingiva
- Nodular mass that protrudes from the interdental papilla, usually in the anterior maxillary region
- Red to pink
- Can be ulcerated
- Typically a small lesion (<2 cm)
- Occurs in younger population (ages 10–19)
- Teeth are typically not affected

TREATMENT

- Local surgical excision down to periosteum
- Remove local sources of irritation (e.g., calculus in adjacent teeth)

► ORAL PIGMENTED LESIONS

Amalgam Tattoo

Implantation of amalgam within the oral mucosa occurs frequently. Broken pieces of amalgam can fall into extraction sites, mucosal abrasions can be contaminated with amalgam particles, and amalgam from endodontic retrograde refills can produce pigmentation within the mucosa. Some cultures intentional tattoo intraorally (e.g., anterior maxillary facial gingiva of Ethiopian women), and occasionally tattoos are placed on the labial mucosa of adults in the United States.

CLINICAL SIGNS AND SYMPTOMS

- Black, blue, or gray macules.
- Most commonly affects gingiva, alveolar mucosa, and buccal mucosa.
- Lateral spread of pigmentation can occur several months after implantation.
- Fragments can be seen on dental radiographs.

TREATMENT

No treatment is required if fragments are detected radiographically. If no fragments are detected, then biopsy is needed to rule out melanocytic neoplasia.

Acquired Nevi

The common mole, represents a benign localized proliferation of melanocytes in the epidermis, or rarely, intraorally. They evolve through several stages:

- Junctional:
 - Earliest presentation, appearing as well-demarcated brown/black macule <6 mm.
 - Histologically appear as nests of melanocytes located at the basal cell layer.
- Compound:
 - Slightly elevated, proliferation of nevus cells.
 - Histologically appear as theques or collections of melanocytes in the dermis and junction of the epithelium and connective tissue.
- Intradermal:
 - Later stage, may present with loss of pigmentation, and surface may become papillomatous with hairs. Nests of nevus cells are found in the underlying connective tissue of the skin. Usually still <6 mm.
- Intramucosal:
 - Nests of nevus cells found in underlying connective tissue of the mucosa.

CLINICAL SIGNS AND SYMPTOMS

- Intraoral nevi are not common
- Typically present on palate or gingiva
- Nevi typically remain less than 6 mm in diameter

TREATMENT

- Biopsy of intraoral pigmented lesions is indicated to rule out melanoma.
- Some authors believe that no treatment indicated unless it is cosmetically bothersome, chronically irritated, or changes in size, shape, and/or color.
- Conservative excision if removal is desired.

Blue Nevus

This is a benign proliferation of dermal melanocytes deep within the connective tissue. There are two types, the **common blue nevus** (see Fig. 10–5) and the **cellular blue nevus**. The color blue is due to the fact that light interacts with melanin particles deep to the surface and reflects back a shorter wavelength light (blue), and absorbs longer wavelengths (red and yellow). This is known as the **Tyndall effect**.

CLINICAL SIGNS AND SYMPTOMS

- Can be found on any cutaneous or mucosal site
- Oral lesions typically found on the palate
- Macular or dome-shaped, blue, <1 cm
- Cellular nevus more rare, develops in the 2nd to 4th decades, usually ≥2 cm

TREATMENT

- Malignant transformation is rare
- Can mimic early melanoma, thus biopsy should be done for intraoral pigmented lesions via a conservative surgical excision



FIGURE 10-5. Blue nevus. (Also see color insert)

(Reproduced with permission from, Wolff K et al. Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology. New York: McGraw-Hill, 2005: 173.)

Malignant Melanoma

These are tumors of melanocytes, rarely arising from a preexisting melanocytic nevus. Acute and chronic sun damage is known to be major etiologic factors. Risk factors include: fair complexion, light hair, skin that sunburns easily, a history of painful or blistering sunburns in childhood, personal or family history of melanoma, dysplastic nevi, or congenital nevi. There are four types of melanoma, each with their own clinical signs and symptoms: superficial spreading, nodular, lentigo maligna, and acrolentiginous.

CLINICAL SIGNS AND SYMPTOMS

- Superficial spreading:
 - Most common form of melanoma
 - Radial growth
 - Most common form of melanoma
 - Macular to a slightly raised lesion
- Nodular:
 - Accounts for 15% of cutaneous melanomas
 - Makes up 30% of head and neck melanomas
 - Begins in the vertical growth phase
 - Nodular lesion
 - Deeply pigmented
 - Poor prognosis
- Lentigo maligna:
 - Accounts for 5% of cutaneous melanomas
 - Large, slowly expanding macule with irregular borders
 - Variety of colors
 - Elderly
 - Predilection for midface region

ABCD—increasing malignant potential of nevus

Asymmetry

Border—irregular or notching

Color—varying shades of brown, black, red, white, and/or blue

Diameter—>6 mm in diameter

BANS—Sites with poor melanoma prognosis

Back

Arms (posterior upper)

Neck (posterior and lateral)

Scalp

- Acrolentiginous:
 - Most common form of melanoma in blacks
 - Seen on hard palate, gingiva, or alveolar mucosa
 - Located on hands and soles of feet, nail beds, and mucous membranes
 - Initially macular, then progresses to nodular lesion

TREATMENT

Surgical excision. Prognosis for oral melanoma is poor, often requiring radical excision.

► DISEASES WITH ORAL PIGMENTATION**Peutz-Jeghers Syndrome**

A skin lesion characterized by freckle-like spots on the hands, perioral skin, and oral mucosa. Also connected with intestinal polyposis and increased risk for GI adenomas, ovarian, breast, and pancreatic carcinoma. The polyps themselves are not considered cancerous. The syndrome can be genetically propagated as an autosomal dominant trait.

CLINICAL SIGNS AND SYMPTOMS

- Small blue-gray macules around the vermilion zone, labial, and buccal mucosa and tongue.
- Perioral freckles around the mouth, nose, anus, and genital areas.
- Do not wax and wane with exposure to sunlight.
- Intestinal polyps are benign hamartomatous growths of intestinal glandular epithelium, throughout the mucus-producing areas of the GI tract.

TREATMENT

These patients should be followed by a GI specialist for development of intestinal intussusception and tumor formation.

Addison's Disease

Also known as **primary hypoadrenocorticism**, this is a disease where there is insufficient production of corticosteroids from the adrenal cortex. This is caused by destruction of the adrenal cortex via autoimmune processes, infections (e.g., TB, fungal disease, AIDS), and other destructive processes like metastatic tumors, sarcoidosis, and hemochromatosis.

CLINICAL SIGNS AND SYMPTOMS

- Fatigue, irritability, depression, weakness, and hypotension.
- Diffuse patchy brown macular pigmentation on oral mucosa usually appears first.
- Bronzing and hyperpigmentation of the skin, most prominent on sun-exposed skin, elbows, and knees.
- Pigmentation is caused by stimulation of melanocytes from increased ACTH levels.
- Rapid ACTH stimulation test and measurement of plasma ACTH levels, which are elevated in primary hypoadrenocorticism.

TREATMENT

- Corticosteroid replacement therapy.
- Dosages may need to be increased for Addison's patients undergoing oral surgery to provide for the body's needs during stressful events.

McCune-Albright Syndrome

This syndrome affects approximately 3% of patients with polyostotic fibrous dysplasia. Patients with this syndrome also have cutaneous and oral pigmentation, along with hyperfunction of one or more of the endocrine glands.

CLINICAL SIGNS AND SYMPTOMS

Patients have café au lait pigmentation, which are typically described as well-defined, unilateral, and tanned macules on the thighs and trunk. Pigmented macules may also present on the oral mucosa. The margins are irregular, which is in sharp contrast to macules of neurofibromatosis, which are smooth.

TREATMENT

See treatment for fibrous dysplasia.

ORAL MELANOTIC MACULE

This is an intraoral focal increase in melanin deposition on the oral mucosa.

CLINICAL SIGNS AND SYMPTOMS

- 2:1, F:M
- Most commonly found on the on the lower lip, buccal mucosa, gingiva, and palate
- Usually solitary lesion
- Well-demarcated
- Uniform color
- Usually no change in size

TREATMENT

Lesions on the lips do not require treatment. Although there is no malignant transformation potential for intraoral lesions, this lesion mimics early melanoma, and therefore should be biopsied to confirm the diagnosis.

► RED AND BLUE LESIONS

Erythroplakia

Is a red plaque for which you do not have a clinical diagnosis, and does not resolve in a matter of weeks with or without treatment. An exact diagnosis can only be made after biopsy. Very often, true erythroplakia will be premalignant or malignant lesion on biopsy.

Median Rhomboid Glossitis

This is a congenital disorder characterized by an erythematous patch on the dorsum of the tongue anterior to the circumvallate papillae (see Fig. 10–6). Can be painful or asymptomatic.

Hereditary Hemorrhagic Telangiectasia

Otherwise known as **Rendu-Osler-Weber disease**, this is an autosomal dominant condition characterized by profuse and often recurrent nosebleeds, multiple telangiectasias in the nasal mucosa, face, and distal extremities, and GI bleeding. Oral features include small blanchable reddish-purple papules on the lips, tongue, buccal mucosa, palate, and gingiva.

Hemangioma

This is a benign proliferation of the blood vessels that gradually disappear on their own. They are typically seen at birth and in childhood, with a predilection for females. They occur as flat or raised lesions that are red or blue, typically on the lips, tongue, or buccal mucosa (see Fig. 10–7). There are three subtypes:

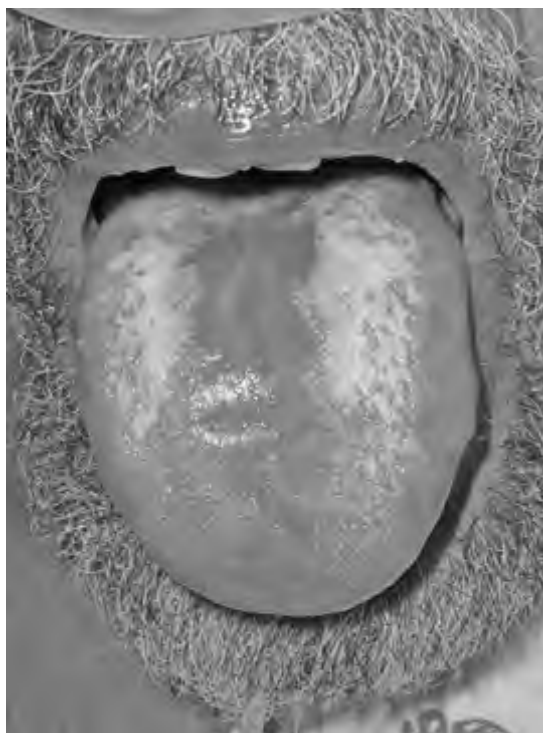


FIGURE 10–6. Median rhomboid glossitis. (Also see color insert)

Note well-demarcated rectangular depapillated area in the midline of the dorsal tongue. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

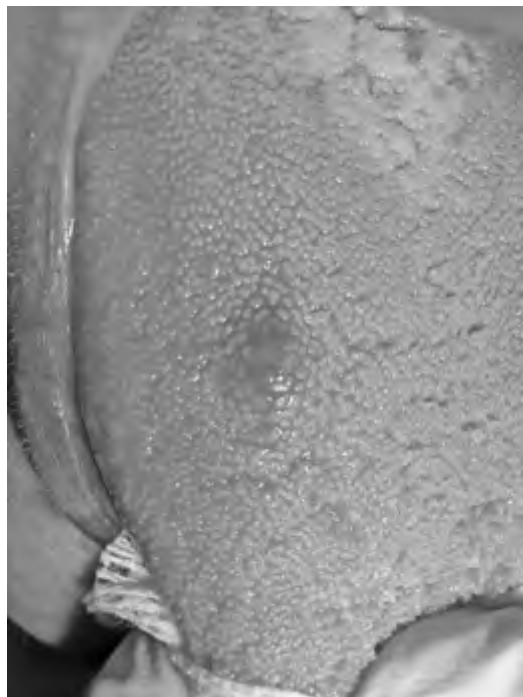


FIGURE 10-7. Hemangioma. (Also see color insert)

Note raised bluish lesion on the dorsal tongue. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

- Capillary:
 - Red-purple
 - Spontaneous involution
 - No bruits or thrills
- Cavernous:
 - Purple-dark blue
 - Involves deeper structures
 - Does not regress
 - No bruits or thrills
- Arteriovenous malformation:
 - Purple-dark blue
 - Bruit and thrill present

TREATMENT

There is usually no treatment required, however larger lesions can be treated with corticosteroid therapy and/or surgical removal. Arteriovenous malformations should not be excised, but rather can be treated with embolization.

► WHITE LESIONS

Leukoplakia

Is a clinical term describing a white patch or plaque, that cannot be rubbed off and for which you do not have a diagnosis. Therefore, leukoplakia is diagnosed only on a clinical basis. Causes of leukoplakia are thought to include tobacco, UV radiation, microorganisms (e.g., *Candida* and *Treponema pallidum*), trauma, and alcohol. Lesions that may present as leukoplakia include:

- Lichen planus
- Frictional keratosis
- Tobacco pouch keratosis
- Nicotine stomatitis
- White sponge nevus
- Leukoedema
- Morsicatio (chronic mucosal cheek trauma)
- Epithelial dysplasia
- Squamous cell carcinoma

CLINICAL SIGNS AND SYMPTOMS

Oral leukoplakia may appear anywhere in the oral cavity. Lesions that show dysplasia or carcinoma frequently occur on the lateral border of the tongue, vermillion border of the lip and the floor of the mouth. Eighty-five percent of oral cancers present clinically as leukoplakia. Males > females. Average age = 60. There are several forms of leukoplakia (see Table 10–4).

DIAGNOSIS

Because leukoplakia is a clinical term, the specific diagnosis can only be made on biopsy. Twenty percent of leukoplakias reveal epithelial dysplasia, a premalignant lesion, or squamous cell carcinoma on histopathologic examination. Those leukoplakias which represent premalignant changes are referred to as epithelial dysplasia. Epithelial dysplasia begins in the basilar and parabasilar areas of the epithelium. Dysplasia is characterized by the following features:

TABLE 10–4. Stages and Clinical Manifestations of Leukoplakia

TYPE OF LEUKOPLAKIA	CLINICAL APPEARANCE
Mild or thin	■ Tends to disappear or continue unchanged ■ Gray, gray-white ■ Translucent ■ Soft ■ Flat ■ Typically show no evidence of dysplasia
Homogeneous or thick	■ Leathery ■ Deepened fissures
Granular or nodular	■ Development of surface irregularities
Verrucous or verruciform	■ Exophytic
Proliferative verrucous	■ Roughened surface ■ Slowly spreading and involving multiple oral mucosal sites ■ 4:1 F:M

- Enlarged nuclei
- Prominent nucleoli
- Increased nuclear:cytoplasmic ratio
- Hyperchromatic nuclei
- Pleomorphic cells and nuclei
- Increased mitotic figures
- Dyskeratosis and keratin pearls
- Loss of cellular cohesion

White Sponge Nevus

Also known as **familial epithelial hyperplasia** or **Cannon's disease**, this is an autosomal dominant genetic disorder caused by a mutation in the keratin 4 and keratin 13 genes. The lesion presents as a bilateral white, rough, surface lesion caused by epithelial thickening of the buccal mucosa. The lesion mimics cheek biting or squamous cell carcinoma. No treatment is required.

Hairy Tongue

Consists of an accumulation of keratin on the filiform papillae of the tongue from a decrease in the keratin desquamation, giving the appearance of a hair on the tongue (see Fig. 10–8). Smoking is the most likely etiologic factor, but other causes include mints, mouth rinses, and candies. It appears on the midline dorsum of the tongue just anterior to the circumvallate papillae. Lateral and anterior regions of the tongue are typically spared. No treatment is required.

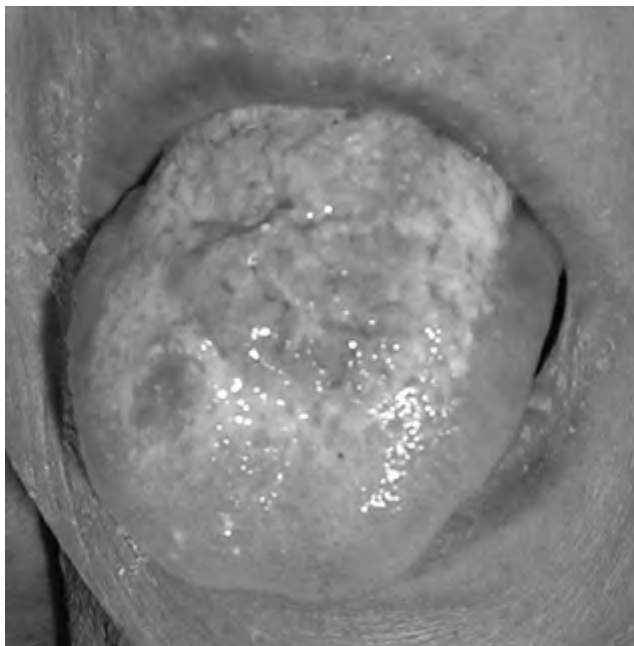


FIGURE 10–8. Hairy tongue. (Also see color insert)

Note yellowish-brown discoloration. Numerous fine hair-like filiform papillae cover the dorsal surface. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

Oral Candidiasis

A common oral fungal infection caused by a yeast-like fungal organism *Candida albicans*. Two types exist, the yeast form and the hyphae form. Candidiasis can present as an opportunistic infection, as a result of clinically induced immunosuppression (e.g., chronic steroid therapy), or as a result of oral flora disturbance (e.g., chronic broad spectrum antibiotic use).

CLINICAL SIGNS AND SYMPTOMS

There are several clinical types of Candidiasis, each with their unique appearance and symptoms (see Table 10–5). Patients can present with one or several clinical subtypes. Diagnosis is based on clinical presentation and exfoliative cytologic exam. Periodic acid-Schiff (PAS) staining and KOH preparation helps identify hyphae and yeasts.

TREATMENT

- Nystatin suspension or lozenge, swish, and swallow
- Imidazole agents:
 - Clotrimazole (troches)
 - Ketoconazole (systemic medication)
- Triazole agents:
 - Fluconazole (systemic medication)
 - Biopsy any remaining lesions after completion of antifungal therapies

TABLE 10–5. Clinical Description of Different Types of Candida Infections

CANDIDA TYPE	APPEARANCE AND SYMPTOMS	ORAL SITES	COMMON ETIOLOGY
Pseudomembranous	Removable, white, cottage cheese-like plaques Erythematous base Burning sensation Foul taste	Dorsum of tongue Buccal mucosa Palate	Immunosuppression (e.g., AIDS, chronic steroid therapy) Antibiotic therapy
Erythematous	Red macules Burning sensation	Posterior hard palate Buccal mucosa Dorsum of tongue	Immunosuppression Antibiotic therapy Xerostomia
Angular cheilitis	Red, fissured lesions Irritated Raw	Commissure of lips	Immunosuppression Loss of vertical dimension Vitamin deficiency
Hyperplastic (candidal leukoplakia)	Nonremovable white plaques Asymptomatic	Anterior buccal mucosa	Immunosuppression Smokers
Central papillary atrophy (median rhomboid glossitis)	Red, atrophic mucosa Asymptomatic	Midline posterior dorsal tongue	Immunosuppression Anatomic
Denture stomatitis	Red Asymptomatic	Mucosa under palatal denture	Denture

Papilloma

A benign epithelial lesion related to the human papilloma virus (HPV), specifically types 6 and 11. They are typically not associated with any malignant transformation.

CLINICAL SIGNS AND SYMPTOMS

Soft, painless lesions, pedunculated with a cauliflower-like surface (verruroid).

TREATMENT

Conservative surgical excision.

Verruca Vulgaris

This is a virally induced squamous proliferation caused by HPV types 2, 4, and 40. It is contagious and spreads via self-inoculation, such as when one puts contaminated fingers into one's mouth.

CLINICAL SIGNS AND SYMPTOMS

Commonly found on the skin and less commonly on the oral mucosa. Typically seen in children.

TREATMENT

Conservative surgical excision.

Verruciform Xanthoma

A hyperplastic condition characterized by lipid-laden histiocytes within the oral cavity. The true etiology is unknown.

CLINICAL SIGNS AND SYMPTOMS

- Frequently located on gingiva and alveolar tissue
- Well-demarcated, soft, painless, sessile, slightly elevated mass
- White, yellow-white, or red
- Papillary or roughened texture
- Usually small (<2 cm)

TREATMENT

Verruciform xanthoma is treated with conservative surgical excision.

Inflammatory Papillary Hyperplasia

Also known as **denture papillomatosis**, this reactive tissue growth develops underneath dentures, typically those that are ill-fitting, or in patients with poor denture hygiene.

CLINICAL SIGNS AND SYMPTOMS

- Usually found on hard palate, directly beneath denture base (see Fig. 10–9).
- Can also be found on mandibular alveolar ridge or on the surface of an epulis fissuratum.
- Mucosa is erythematous and has a pebbly or papillary surface.
- Associated with denture sore mouth.

TREATMENT

- Removal of denture may allow lesion to subside.
- Excision of advanced or collagenized lesions.
- Cryosurgery.
- Curettage.

Inflammatory Fibrous Hyperplasia

Also known as **epulis fissuratum**, this is a tumor-like hyperplasia of fibrous connective tissue, frequently associated with the flange of an ill-fitting denture.

CLINICAL SIGNS AND SYMPTOMS

- Presents as folds of hyperplastic tissue in the alveolar vestibule, typically on the facial aspect of the alveolar ridge
- Can appear erythematous and ulcerated
- Size can vary

TREATMENT

Surgical removal of the epulis. Ill-fitting dentures should be remade or relined.

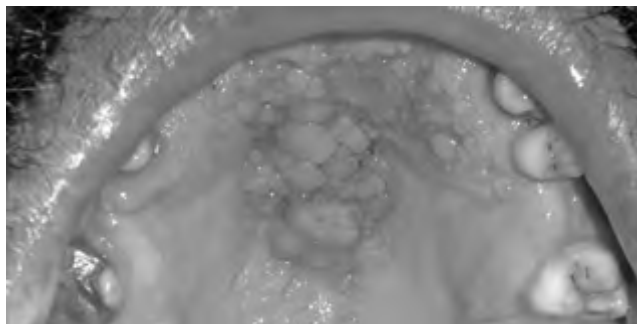


FIGURE 10–9. Inflammatory papillary hyperplasia. (Also see color insert)

Papillary change of the hard palate in a patient who continuously wore his partial denture. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

Eruption Cyst

This is a soft tissue analogue to the dentigerous cyst and develops from around the crown of an erupting tooth that is within the soft tissues overlying the alveolar bone.

CLINICAL SIGNS AND SYMPTOMS

- Soft, translucent swelling in gingival mucosa.
- Associated with erupting primary or permanent tooth.
- Commonly seen in the mandibular molar area.
- When traumatized, the lesion can appear purplish or brown.

TREATMENT

No treatment required as the cyst typically ruptures on its own. Simple excision of the roof of the cyst can accelerate the eruption process.

Dentigerous Cyst

This is the most common type of odontogenic cyst. It encloses the crown of an unerupted tooth and is attached to the tooth at the cemento-enamel junction. It is a cyst that originates by the separation of the follicle from around the crown of an unerupted tooth. Some cysts develop earlier in odontogenesis and result in hypoplastic teeth.

CLINICAL SIGNS AND SYMPTOMS

- Mostly involves mandibular third molars, followed by maxillary canines.
- Frequently found in younger patients (ages 10–30).
- Small cysts are asymptomatic.
- Large cysts can be associated with painless expansion of the bone.
- More extensive lesions can cause facial asymmetry.
- Some can become infected, swollen, and painful.
- Unilocular radiolucency associated with an unerupted tooth, well-defined sclerotic border.
- *Central* type: cyst surrounds crown.
- *Lateral* type: associated with mesioangularly impacted mandibular third molars. Cyst grows laterally along root surface, partially surrounding crown.
- *Circumferential* type: surrounds crown and extends along root.
- Maxillary teeth can be displaced to the floor of the nose. Mandibular teeth can be displaced to inferior border of mandible.
- Root absorption in adjacent teeth.
- Radiography not diagnostic, since other odontogenic/nonodontogenic tumors resemble those of dentigerous cyst.
- Potential for epithelial lining to undergo neoplastic transformation to ameloblastoma, squamous cell carcinoma, or intraosseous mucoepidermoid carcinoma.

TREATMENT

- Enucleation of cyst and removal of associated tooth.
- Occasionally, orthodontic treatment can help with tooth eruption.
- Large cysts can be treated initially with marsupialization and excision later.

Odontogenic Keratocyst (OKC)

A cyst arising from the cell rests of the dental lamina, OKCs are benign cystic neoplasms. Growth of these cysts is related to factors inherent in the epithelium or enzymatic activity in the fibrous wall.

CLINICAL SIGNS AND SYMPTOMS

- OKCs are lesions that affect men more than women.
- Small OKCs are asymptomatic.
- Larger OKCs associated with pain, swelling, or drainage.
- May grow in medullary cavity of bone without causing obvious bone expansion.
- Radiographically, OKC is a well-defined radiolucent lesion with smooth and often corticated margins. It may be uni- or multilocular.
- Resorption of roots in OKC less common than in dentigerous and radicular cysts.
- Multiple OKCs seen in **nevroid basal cell carcinoma (Gorlin) syndrome**.

TREATMENT

- Enucleation and curettage
- Tends to recur
- Similar risk to other cysts for malignant transformation

Nevroid Basal Cell Carcinoma Syndrome

Also known as **Gorlin syndrome**, this is an autosomal dominant condition, with specific skin and bone manifestations.

CLINICAL SIGNS AND SYMPTOMS

- Multiple basal cell carcinomas on cheeks, upper eyelids, nose, and trunk
- Palmar and plantar pits
- Hyperkeratosis of palms and soles
- Epidermal cysts, lipomas, and fibromas
- Multiple OKCs of the jaws with high recurrence rates
- Bifid ribs
- Kyphoscoliosis
- Calcification of the falx cerebri

TREATMENT

- Jaw cysts treated by enucleation.
- Most anomalies in nevroid basal cell carcinoma are not life-threatening.

Periapical Cyst

Also known as **radicular cysts**, these lesions are stimulated by inflammatory reactions to form a true epithelium-lined cyst at the apex of a nonvital tooth. Sources of epithelial cells are derived from the rests of Malassez, fistulous tracts, crevicular epithelium, or sinus lining.

CLINICAL SIGNS AND SYMPTOMS

- Usually asymptomatic unless there is an acute inflammatory exacerbation. Large cysts may swell and produce sensitivity.
- Movement and mobility of adjacent teeth are possible as the cyst enlarges.
- The involved tooth doesn't respond to thermal or electric pulp testing.
- Small periapical radiolucencies and loss of lamina dura along adjacent roots are observed on periapical or panoramic radiographs.

TREATMENT

- Similar treatment to periapical granulomas
- If there is evidence of periapical inflammation, extraction or root canal therapy of the tooth is recommended
- Large lesions can be treated with endodontic therapy, biopsy, and marsupialization
- Follow up at 1 to 2 year intervals to rule out any enlargement of the lesion is recommended
- If the lesion fails to resolve after root canal therapy or if the endodontically treated tooth shows an expanding lesion, periapical surgery and biopsy are indicated to rule out other possible pathologic processes
- Cysts that fail to resolve should be surgically excised, since they appear similar to some odontogenic and nonodontogenic tumors

► NONODONTOGENIC CYSTS

Developmental cyst

- Nasolabial cyst:
 - Originally thought to be fusion of the globulomaxillary processes.
 - True origin remains in doubt
 - May be derived from remnants that form nasolacrimal duct
 - Presents as swelling in lateral aspect of upper lip, high up in the sulcus
 - Treatment: Local excision
- Median alveolar cyst:
 - Also known as the median mandibular cyst
 - Originally thought to be a cystic lesion formed at the line of fusion of each half of the mandibular arch
 - Now thought to be another type of odontogenic cyst or tumor
- Globulomaxillary cyst:
 - Originally thought to be a fissural cyst caused by retained epithelial remnants at the fusion of the maxillary process with the globular process
 - Pear-shaped well-circumscribed radiolucency in the maxilla between lateral incisor and canine
 - Teeth are vital
 - Currently not thought to be fissural cyst, but rather reclassified as OKC < radicular cysts, central giant cell granulomas, calcifying odontogenic cysts, and odontogenic myxomas based on histologic findings

- Nasopalatine duct cyst:
 - Also known as incisive canal cyst
 - True fissural cyst
 - Soft swelling behind upper anterior teeth
 - Thought to be derived from epithelial remnants of the paired embryonic nasopalatine
 - Males > females
 - Well-circumscribed radiolucency in the midline of the anterior palate
 - Needs to be distinguished from nasopalatine foramen
 - Treatment: Local curettage
- Median palatal cyst:
 - Rare developmental cyst located in the midline of the hard palate
 - Needs to be distinguished from nasopalatine foramen
 - True fissural cyst
 - Considered a posteriorly placed nasopalatine duct cyst

Congenital Cyst

- Thyroglossal duct cyst:
 - Considered to be remnants of the thyroglossal tract, which runs from foramen cecum to the thyroid gland
 - Midline of neck or to one side
 - Floor of mouth, tongue, or near the thyroid cartilage
- Branchiogenic cyst:
 - Also known as brachial cleft cyst
 - Originates from transformation of salivary gland tissue which is present in the cervical lymph nodes
 - Entrapment of epithelium in the branchial apparatus is the most accepted theory
 - Presents as well-circumscribed, freely movable mass along the anterior border of the sternocleidomastoid muscle
- Dermoid and Epidermoid cysts:
 - Epithelium trapped in connective tissue during embryogenesis
 - Clinically presents as well-circumscribed, compressible soft tissue enlargement found in the floor of the mouth, submandibular areas, and sublingual areas
 - Dermoid form has sebaceous glands, hair follicles, and sweat glands
 - Treatment: Enucleation
 - Recurrence is rare

► PSEUDOCYSTS

Traumatic Bone Cyst

Also known as idiopathic bone cyst, simple bone cyst, and latent bone cyst, this lesion is thought to be the result of intramedullary hemorrhage from trauma. Instead of formation of new bone in the area of injury, the blood clot liquefies and leaves an empty space.



Traumatic bone cysts scalloped margins between roots of teeth.

CLINICAL SIGNS AND SYMPTOMS

- Asymptomatic
- Commonly in the posterior mandible
- Well-circumscribed radiolucency with a scalloped margin beneath the tooth roots

TREATMENT

- Surgical exploration and curettage
- Provides for a specimen to rule out other lesions (e.g., OKC) and promotes healing

Aneurysmal Bone Cyst

This is a lesion of the jaw that contains blood filled spaces surrounded by fibroblastic connective tissue containing giant cells and osteoid trabeculae.

CLINICAL SIGNS AND SYMPTOMS

- Radiographically, appears as a well-circumscribed soap bubble lesion.
- Clinically presents as mandibular expansion or ballooning, typically in those 30 years and younger. Often compared to a “blood soaked sponge.”

TREATMENT

- Responds well to aggressive curettage. Recurrences are rare.

► VESICULOBULLOUS DISEASE

Aphthous Ulcers

These are common painful lesions with periodic recurrences, sometimes also referred to as **recurrent aphthous stomatitis**. There are several theories regarding the etiology of aphthous ulcers including allergic reactions, genetic predisposition, hematologic abnormalities, infectious agents, nutritional imbalances, stress, trauma, or hormonal influences. The lesions are typically limited to nonkeratinized mucosa (Fig. 10–10).

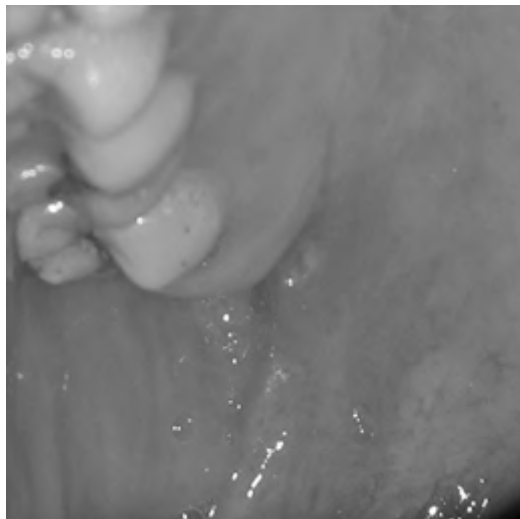


FIGURE 10–10. Aphthous ulcer. (Also see color insert)

Ulcer of the far right soft palate with a bright red halo. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

CLINICAL SIGNS AND SYMPTOMS

- Recurrent aphthous major:
 - Approximately 1–3 cm in diameter
 - Heal in 2–6 weeks with possible scarring
 - Deeper lesions compared with minor type
- Recurrent aphthous minor:
 - Begins as red macule
 - Develops into a 3–10 mm ulceration
 - Removable membrane with an erythematous halo
 - Heals in 1–2 weeks without scarring
- Recurrent herpetiform:
 - Clinically similar to primary HSV infection
 - Large numbers of lesions
 - High recurrence rate
 - Any mucosal surface may be involved

TREATMENT

Topical and systemic corticosteroids.

Pemphigus vulgaris

There are several types of pemphigus. The most common type is pemphigus vulgaris. Pemphigus vegetans is seen in the oral cavity on rare occasions. They belong to a group of immune mediated vesiculobullous diseases.

CLINICAL SIGNS AND SYMPTOMS

- Typically seen in adults.
- Male = Female.
- Initial lesions are vesicles or bullae (see Fig. 10–11).
- Blisters are fragile and rupture forming painful ulcers that heal slowly.



FIGURE 10–11. Pemphigus vulgaris. (Also see color insert)

Vesiculobullous lesions of the vermillion border of the lip. Note intact vesicle (white area with red halo) on the right buccal mucosa. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

- Nikolsky's sign: rubbing of uninvolved mucosa creates a blister.
- Oral lesions often start prior to skin involvement.
- Histological presentation:
 - Intraepithelial cleft formation.
 - Basal cells remain attached to basement membrane creating what appears as a row of tombstones on the basement membrane.
 - Tzanck cells: acantholytic epithelial cells with enlarged dark nuclei.
 - Direct immunofluorescence: IgG and IgM autoantibodies.
 - Autoantibodies are directed against components of desmosomes, which are responsible for bonding epithelial cells together.

TREATMENT

- Aggressive steroid treatment and other immunosuppressive drugs like azathioprine are used. Can be fatal if not treated.

Pemphigoid

There are several types of pemphigoid, all caused by autoantibodies that target a component of the basement membrane (Fig. 10–12). Benign mucous membrane pemphigoid (also known as cicatricial pemphigoid) is the form seen in the oral cavity.

CLINICAL SIGNS AND SYMPTOMS

- Vesicles and ulcerations involving the skin, conjunctiva of the eye, nose, pharynx, esophagus, and larynx.
- Nikolsky's sign may be present.
- Gingival involvement with diffuse erythema.
- *Cicatricial type*:
 - Vesicles and ulcers that heal with scarring outside of the oral cavity. No scarring is seen in the oral cavity.
 - Ocular involvement:
 - Subconjunctival fibrosis
 - Adhesions
 - Scarring may lead to blindness



FIGURE 10–12. Pemphigoid. (Also see color insert)

(Reproduced with permission from, Wolff K et al. Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology. New York: McGraw-Hill, 2005: 109.)

- Histological presentation:
 - Subepithelial cleavage from the underlying connective tissue. The basal cells remain attached to the epithelium.
 - Direct immunofluorescence shows continuous linear band of IgG and C3 along the basement membrane.

TREATMENT

Topical and/or systemic corticosteroids. Ophthalmology consult for cicatricial type to rule out the presence of ocular involvement.

Lichen Planus

This disease is caused by cytotoxic cell-mediated hypersensitivity. Some evidence to suggest an association of hepatitis C, with virally induced antigen stimulation. There are two clinical forms of lichen planus, reticular and erosive. Histologically, they present with degeneration of the basal cell layer, hyperkeratosis, “band-like” lymphocytic infiltration adjacent to epithelium, “saw tooth” rete ridges, and Civatte bodies.

CLINICAL SIGNS AND SYMPTOMS

- Reticular:
 - Most common type
 - Asymptomatic
 - White lesions due to epithelial thickening (Wickham’s striae)
- Erosive (see Fig. 10–13):
 - Painful ulcerations
 - Erythematous gingiva

TREATMENT

- No treatment required for asymptomatic lesions.
- Topical or systemic steroids for symptomatic lesions.



FIGURE 10-13. Erosive lichen planus. (Also see color insert)

Note reticular striated areas of the right buccal mucosa with associated erosions. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

Erythema Multiforme

This is considered to be a vesiculobullous disease which is reactive in nature. Patients may have skin lesions that present mainly on the extremities and face; however oral lesions can also present as vesicles and ulcerations. Typical causes include drugs (e.g., sulfa drugs), vaccinations, and viral infections (e.g., HSV). Erythema multiforme is divided into erythema multiforme minor and erythema multiforme major (Stevens-Johnson syndrome). Toxic epidermal necrolysis is a more severe manifestation of erythema multiforme.



Erythema multiforme–target lesions.

Clinical Signs and Symptoms

- Target lesions on extremities and face.
- Wide spread oral vesicles and ulcers on buccal mucosa (see Fig. 10–14). Typically symmetric.
- Oral lesions may exist without skin lesions.
- Crusting and bleeding of lips.
- Erythema multiforme minor is most often triggered by HSV.
- *Stevens–Johnson syndrome*:
 - More severe form of erythema multiforme
 - Triggered by drugs
 - Lesions on skin, oral mucosa, conjunctiva, and genitalia
- *TEN*:
 - Most severe form of erythema multiforme
 - Female predominance
 - 1/3 mortality rate

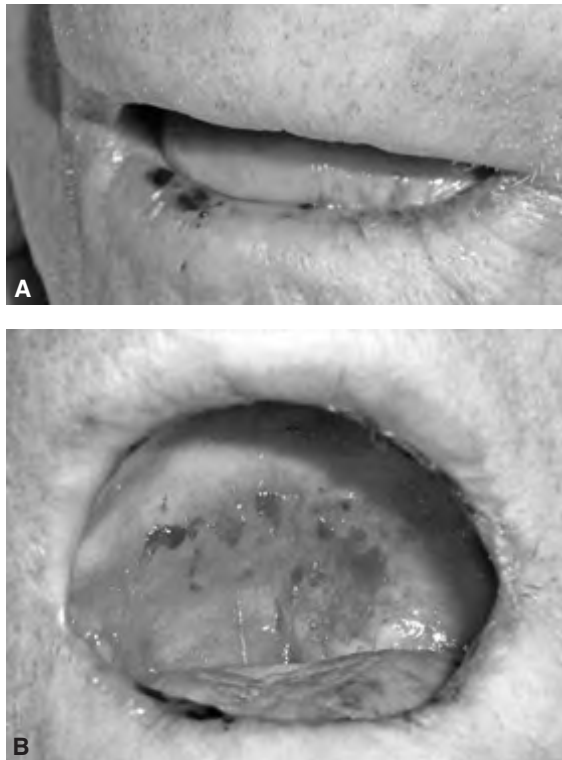


FIGURE 10–14. Erythema multiforme. (Also see color insert)

A. Crusted lesions of the lower lip. B. Numerous hemorrhagic erosions of the soft palate.
(Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

TREATMENT

Erythema multiforme minor is treated with topical or systemic corticosteroids, and/or acyclovir. Any causative drugs should also be removed if possible. Stevens–Johnson syndrome is treated with steroids and also with removal of the offending drug. TEN requires treatment in a hospital burn unit.

► INFECTIOUS VESICULOBULLOUS DISEASES

Group A coxsackievirus

The two most important and common group A coxsackievirus manifestations are **herpangina** and **hand, foot, and mouth disease**.

CLINICAL SIGNS AND SYMPTOMS

- Herpangina:
 - Vesicles and ulcers on soft palate, uvula, and anterior tonsillar pillar
 - Typically resolves in several days
 - Fever, sore throat, nausea, vomiting, diarrhea, and lymphadenopathy
- Hand, foot, and mouth disease:
 - Vesicles and ulcers throughout the oral cavity
 - Macules and vesicles on the hands and feet
 - Oral lesions resemble herpangina, but can be larger

TREATMENT

- Symptomatic treatment
- Typically resolves on its own after several days

Acute Herpetic Gingivostomatitis

Also known as primary herpes, these lesions are caused by the herpes simplex virus 1 or 2 (more commonly HSV 1). The lesion occurs in both children and adults.

CLINICAL SIGNS AND SYMPTOMS

- Acute onset of malaise, fever, and lymphadenopathy.
- Multiple vesicles and ulcers in oral cavity.
- Gingival swelling and erythema (see Fig. 10–15).
- Can remain latent in regional ganglia.
- Reactivation of latent viruses can occur with UV exposure, trauma, or immunosuppression.
- **Recurrent herpetic stomatitis:**
 - Vesicles and ulcers occurring in small clusters
 - Lip, gingiva (attached mucosa), and hard palate
 - Preceded by prodrome of tingling, pain, or numbness in the area of the vesicles
- **Herpes labialis**
 - Otherwise known as secondary herpes
 - Caused by the same viruses



FIGURE 10-15. Acute herpetic gingivostomatitis. (Also see color insert)

Note the erythematous, inflamed gingiva with vesicular lesions on vermilion border and anterior tongue. The child clinically had fever and lymphadenopathy. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

TREATMENT

- Symptomatic treatment
- Acyclovir, when initiated early in the course of primary or recurrent herpes
- Usually resolves on its own in 10–14 days

Varicella Zoster

Caused by the varicella-zoster virus (VZV), otherwise known as varicella, or chickenpox. There are two manifestations of the VZV, a primary infection (varicella), and the recurrent infection (Zoster or shingles), which typically occurs in the elderly and those who are immunocompromised.

CLINICAL SIGNS AND SYMPTOMS

- Primary infection:
 - Prodromic malaise, fever, and lymphadenopathy
 - Macules, papules, vesicles, and ulcers on skin and oral mucosa (see Fig. 10-16)
 - Pruritic skin lesions
- Recurrent infection:
 - Painful vesicles, ulcers and crusts following a dermatomal distribution
 - Commonly seen on trunk and trigeminal area
 - Unilateral, unless immunocompromised

TREATMENT

- Supportive therapy.
- Avoid ASA in children because of Reye's syndrome.
- Vaccine is available for varicella zoster virus.



FIGURE 10-16. Varicella Zoster (Herpes Zoster). (Also see color insert)

Crops of vesicles located antero-posteriorly along the buccal mucosa. Note crusted lesions on the right commissure that extended onto the skin of the right cheek. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

► INFECTIOUS DISEASES

Actinomycosis

A microbiological infection caused by *Actinomyces* species (e.g., *A. israelii*, *A. naeslundii*, *A. viscosus*, *A. odontolyticus*, *A. meyeri*, and *A. bovis*), which are gram positive anaerobic bacteria.

CLINICAL SIGNS AND SYMPTOMS

- Wooden induration with a central soft spot
- Sulfur granules in pus
- Spread of infection does not follow fascial planes and lymph channels

TREATMENT

Six- to twelve-week course of antibiotics (e.g., penicillin or tetracycline).

Histoplasmosis

The most common systemic fungal infection caused by *Histoplasma capsulatum*. Endemic to Mississippi–Ohio–Missouri River basin. The infection is caused by soil exposure in humid areas that is contaminated by birds and bats.

CLINICAL SIGNS AND SYMPTOMS

- Coin lesion on chest X-ray
- Clinically similar to tuberculosis
- Gingival manifestations: Chronic proliferative granulomatous tissue lesions, which can be painful or asymptomatic

TREATMENT

Amphotericin or imidazoles.

Syphilis

A sexually transmitted infectious disease caused by the organism *Treponema pallidum*, via direct contact or vertical transmission.

CLINICAL SIGNS AND SYMPTOMS

- Primary stage:
 - A painless ulcer (chancre) occurs at 3 weeks
 - Spontaneously heals
 - Contagious
- Secondary stage:
 - 4–10 weeks after infection
 - Maculopapular rashes
 - Mucous patches
 - Condyloma lata: Papillary lesions that resemble viral papillomas
 - Lues maligna: Seen in immunocompromised patients
 - Spontaneously heals in 3–12 weeks
 - Contagious
- Tertiary stage:
 - Develops after a latent period (1–20 years)
 - Gumma develop, which are large tissue ulcerations
 - Leutic glossitis: Atrophy and loss of dorsal tongue papillae
 - CNS manifestations (neurosyphilis):
 - Psychosis
 - Dementia
 - Death
 - Noncontagious
- Congenital syphilis:
 - Characterized by Hutchinson's triad:
 - Hutchinson's teeth: Screwdriver-like incisors, mulberry molars
 - Interstitial keratitis (inflammation of the corneal stroma)
 - 8th nerve deafness
 - Saddle nose deformity
 - High arched palate
 - Frontal bossing
 - Short maxilla



Syphilis–Hutchinson's triad

- Hutchinson's teeth
- Corneal inflammation
- Deafness

TREATMENT

- Primary and secondary stage:
 - Benzathine penicillin IM
 - Tetracycline or doxycycline for 14 days for patients with penicillin allergies
 - Latent infections should be treated with penicillin once weekly for 3 weeks
 - Neurosyphilis treated with intravenous (IV) penicillin

Traumatic Neuroma

A benign soft tissue neoplasm associated with trauma to a peripheral nerve, such as pressure by a denture against the mental foramen or transection of a nerve bundle. Typically presents as smooth-surfaced, nonulcerated nodules with pain.

Multiple Endocrine Neoplasia

Associated with an autosomal-dominant pattern of inheritance, or spontaneous mutation, with three subtypes:

MEN I "P" organs:

Pancreas
Pituitary
Parathyroid

- MEN type I:
 - Pancreatic, parathyroid, and pituitary tumors
- MEN type II (IIa):
 - Medullary carcinoma of the thyroid, pheochromocytoma, parathyroid gland hyperplasia
- MEN type III (IIb):
 - Pheochromocytoma
 - Oral and intestinal ganglioneuromatosis (mucosal neuromas)
 - Medullary carcinoma of the thyroid
 - Usually presents in those 18–25 years
 - Higher propensity for metastasis; death by average age of 21
 - Marfanoid habitus

Neurilemoma (Schwannoma)

A benign encapsulated soft tissue neoplasm that originates from Schwann cells. Appears as a raised, freely movable nodule on sympathetic, peripheral, or cranial nerves, particularly the eighth cranial nerve. Histologically characterized by Antoni A (organized spindle cells forming Verocay bodies) and Antoni B tissue (spindle cells that are randomly arranged).

Neurofibroma

A common peripheral nerve neoplasm, which arises from Schwann cells and perineural fibroblasts. They may exist as solitary lesions or as part of neurofibromatosis type I or von Recklinghausen's disease.

CLINICAL SIGNS AND SYMPTOMS

Neurofibromas arise as solitary tumors in young adults and are slow-growing, soft, painless lesions, varying in size from small nodules to larger masses. The tongue and buccal mucosa are the most common intraoral sites. Rarely, the tumor arises in bone, producing a well-demarcated or poorly defined unilocular or multilocular radiolucency.

TREATMENT

- Localized excision
- Mandibular resection advocated by some
- 5–15% malignant transformation to neurogenic sarcoma in the setting of neurofibromatosis

Neurofibromatosis Type I (von Recklinghausen's Disease)

This is an autosomal dominant syndrome affecting chromosome 17 with cutaneous manifestations. There are several forms of neurofibromatosis, but the most common is type I, otherwise known as von Recklinghausen's disease.

CLINICAL SIGNS AND SYMPTOMS

- Multiple well-circumscribed raised nodules (neurofibromas) on the face, trunk, and extremities
- Neurofibromas can also be found intraorally
- Café-au-lait spots: Multiple brown macules on the skin typically measuring >1.5 cm in adults, and >0.5 cm in children
- Axillary freckling
- Lisch nodules in the iris
- Possible malignant transformation

Fibroma

These are common benign soft tissue neoplasms commonly found on the buccal mucosa and lower lip. They represent hyperplastic fibrous tissues. Clinically appear as firm asymptomatic nodules. No treatment is required.

Congenital Epulis of the Newborn

This rare benign neoplasm has an unknown origin. The lesion is typically seen on the alveolar ridge of the maxilla in newborns.

CLINICAL SIGNS AND SYMPTOMS

- Pedunculated lesion that can grow to be quite large
- Pink-red smooth surface
- Lateral to the midline in the area of the developing incisor and canine
- Females > males

TREATMENT

- Surgical excision with no recurrence.
- Tumor stops growing after birth and may even diminish in size.

Lipoma

A benign soft tissue tumor of fat tissue. Lipomas are typically observed in adults.

CLINICAL SIGNS AND SYMPTOMS

Clinically these lesions are seen on the buccal mucosa, tongue, and the floor of mouth. Outside of the mouth, they appear on the trunk and proximal portions of the extremities. Can appear sessile or pedunculated yellow nodules and soft to palpation.

TREATMENT

Conservative local excision. Recurrence is rare.

Osteoma

This is a benign tumor consisting of mature compact or cancellous bone. The *periosteal* type arises from the surface of bone, while the *endosteal* type arises centrally from within the bone. *Gardner's syndrome* is an autosomal dominant condition characterized by multiple osteomas in the jaws.

CLINICAL SIGNS AND SYMPTOMS

- Males > females
- Asymptomatic
- Slow-growing bony masses
- Radiographically presents as well-circumscribed sclerotic radiopaque masses
- **Gardner's syndrome:**
 - Intestinal polyposis, with nearly 100% malignant potential, requiring colectomy
 - Multiple endosteal osteomas of the jaws, particularly at the angle of the mandible
 - Osteomas also found in facial bones and long bones
 - Fibromas of the skin
 - Epidermal cysts
 - Impacted teeth
 - Odontomas

TREATMENT

- Surgical excision.
- Asymptomatic lesions can be followed clinically and radiographically without treatment.
- Recurrence is rare.

Central Giant Cell Granuloma (CGCG)

Whether or not CGCG is a true tumor or a reactive bone lesion is controversial. CGC lesions show similarities to many benign and malignant lesions, such as giant cell tumors of the long bones and brown tumors of hyperparathyroidism.

However there is some controversy in the literature as to whether or not true giant cell tumors occur in the jaws. True giant cell tumors in the jaw have not been well established. In fact, only one example of malignant giant cell tumor of the jaws has been reported.

CLINICAL SIGNS AND SYMPTOMS

- Mostly asymptomatic and first come to attention during routine radiographic examination.
- Nonaggressive lesions: Present with few or no symptoms, slow growth, do not show root resorption or cortical perforation, low rates of recurrence.

- Aggressive lesions: Characterized by pain, rapid growth, root resorption, and cortical perforation. Tendency to recur.
- Radiographically presents as unilocular or multilocular, well-circumscribed, radiolucency usually found in the anterior mandible.

TREATMENT

- Surgical:
 - Curettage
 - 15–20% recurrence rate
 - Aggressive types require mandibular resection and reconstruction
- Nonsurgical:
 - Intralesional steroid injections
 - Calcitonin subcutaneous injections
 - α -Interferon subcutaneous injections

► FIBROUS DISEASES

Ossifying Fibroma

This is a benign fibro-osseous neoplasm consisting of a cellular fibrous stroma containing varying amounts of calcified tissue.

CLINICAL SIGNS AND SYMPTOMS

- Well-circumscribed mixed density lesion with smooth and sclerotic borders
- Third and fourth decades of life
- Slow growing
- Asymptomatic
- Female > male

TREATMENT

- Tumor enucleation.
- Larger lesions, with bone destruction may require surgical removal with bone grafting.
- Prognosis is generally good, with rare chance of recurrence, and low malignant potential.

Fibrous Dysplasia

This is a benign fibro-osseous disease with an unknown etiology. Fibrous dysplasia can be categorized into four types:

- Monostotic:
 - Involving one bone.
- Polyostotic:
 - Involving two or more bones.
 - When 3/4 of the skeleton is involved, this is referred to as the *Jaffe type*.
- Albright's syndrome:
 - Multiple lesions.
 - Hyperpigmentation.
 - Endocrine disturbances: Precocious puberty and/or hyperthyroidism.



Fibrous dysplasia—ground glass appearance.

- Craniofacial:
 - Confined to bones of the craniofacial complex (zygoma, maxilla, base of skull, sphenoid, frontal bones, nasal bones).

CLINICAL SIGNS AND SYMPTOMS

- Commonly affects the jaws.
- Painless swelling (see Fig. 10–17A).
- Radiographic ground glass appearance, poorly defined (see Fig. 10–17B).
- Expansion can cause compression nerves and blood vessels.
- Maxilla > mandible.
- Mandibular form does not cross the midline.
- Females > males.
- Inactive lesions are asymptomatic.
- Active lesions appear hot on bone scan.
- Occasional tooth displacement.



FIGURE 10–17. Fibrous dysplasia. (Also see color insert)

A. Left maxillary alveolus enlarged in the buccal-lingual dimension. B. Ground glass radiographic appearance of the bone. (Figure B courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

TREATMENT

Biopsy can be done to confirm the lesion, however, the lesion classically burns out on its own in the patient's late teens or twenties. Surgical recontouring is reserved for symptomatic or cosmetically unacceptable lesions during the quiescent phase of the disease. Regrowth occurs in 25–50% of surgically recontoured patients.

Cemento-Osseous Dysplasia (COD)

Considered to be the most common form of fibro-osseous disease, cemento-osseous dysplasia is a disorder in the production of bone and cementum-like tissue in the tooth-bearing areas of the jaws. The etiology is unclear, however local trauma (e.g., abnormal occlusal forces) has been suggested.

CLINICAL SIGNS AND SYMPTOMS

There are three types of COD:

- Periapical COD:
 - Radiolucent, radiopaque, or mixed radiolucent well-circumscribed lesions in periapical areas
 - Associated with vital teeth
 - Anterior mandible
 - African American females
- Focal COD:
 - Asymptomatic
 - Nonexpansile radiolucencies with associated opacities
 - Associated with edentulous areas
 - Mandible
 - May represent abnormal healing after extractions
 - <2 cm
- Florid COD:
 - African American females
 - Painless nonexpansile lesion
 - Two or more jaw quadrants
 - Radiographically presents as multiple confluent lobular radiopacities in tooth-bearing areas
 - Cortical expansion absent

TREATMENT

Limited to surgical recontouring for symptomatic and/or cosmetically unacceptable lesions.

► DISEASES OF THE BONE

Langerhans Cell Disease

Langerhans cells are dendritic cells in the skin and mucosa and serve as antigen presenting cells. Langerhans cell histiocytosis was formerly known as Histiocytosis X, which was further subcategorized as eosinophilic granuloma, Letterer–Siwe disease, and Hand–Schüller–Christian disease. Eosinophilic granuloma is considered the chronic localized form. Letterer–Siwe disease is the acute disseminated form. Hand–Schüller–Christian disease is the chronic disseminated form. The precise etiology is unknown.



Hand-Schüller-Christian disease

1. Punched-out bone lesions
2. Exophthalmos
3. Diabetes insipidus
4. Floating teeth

CLINICAL SIGNS AND SYMPTOMS

- Eosinophilic granuloma:
 - Cupped-out radiolucency resembling periodontal disease (see Fig. 10–18)
 - Affects the jaw in 10–20% of all cases (mandible > maxilla)
- Letterer-Siwe disease:
 - Young children
 - Affects skin, bones, internal organs (lungs and liver)
 - Frequently fatal
- Hand-Schüller-Christian disease:
 - More common in males
 - Punched-out bone lesions in skull and jaws
 - Exophthalmos
 - Diabetes insipidus
 - Second to third decades of life
 - Radiolucencies associated with apices of teeth, appearing as “floating teeth”

TREATMENT

- Eosinophilic granuloma:
 - Aggressive local curettage
 - Intralesional steroid injections
 - Low-dose radiotherapy
- Letterer-Siwe disease:
 - Chemotherapy
- Hand-Schüller-Christian disease:
 - Local curettage
 - Chemotherapy for aggressive forms
 - Low-dose radiation therapy for isolated lesions

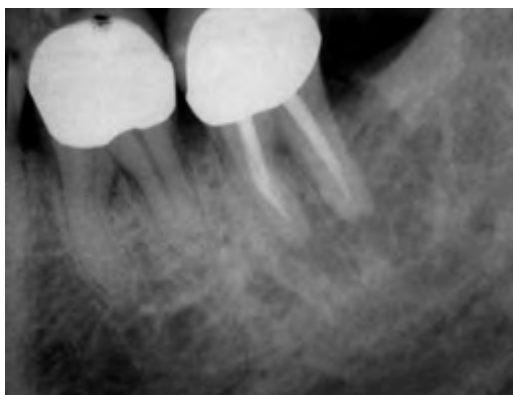


FIGURE 10–18. Eosinophilic granuloma (Langerhans cell disease).

Note the relative radiolucency surrounding an endodontically-treated second molar tooth.
(Courtesy of Drs. Jerald Friedman and John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

Multiple Myeloma

A disorder characterized by proliferation of neoplastic plasma cells. It consists of individual tumors known as plasmacytomas. The plasma cells are monoclonal and produce a specific immunoglobulin heavy or light chain.

CLINICAL SIGNS AND SYMPTOMS

- >50 years of age
- Blacks > whites
- Male > female
- Symptoms of anemia
- Frequent infections with *Streptococcus pneumoniae* and *Haemophilus influenzae*
- Bone pain
- Renal insufficiency
- Pathologic fractures
- Amyloidosis
- Radiographically appears as multiple punched-out radiolucencies and soap bubble appearance
- Lab findings: Serum and urine protein electrophoresis reveals monoclonal gammopathy, most commonly IgG, and free kappa and lambda light chains (**Bence Jones proteinuria**)
- Bone marrow biopsy: 10–20% plasma cells (normal <5%)

TREATMENT

- Thalidomide
- Chemotherapy
- Autologous stem cell transplantation for young patients

Squamous Cell Carcinoma

Squamous cell carcinomas make up the majority of oral cancers. The remainder includes salivary gland adenocarcinoma, metastatic tumors, sarcomas, melanomas, and lymphomas. Alcohol and tobacco are causative factors in the etiology of squamous cell carcinomas. They have a synergistic effect. UV radiation is associated with carcinomas of the lower lip. Human papillomavirus types 16, 18, 31, and 33 are considered the viral types implicated in the etiology of some oral squamous cell carcinomas. Other predisposing factors may include vitamin deficiency, immunocompromised status, and iron deficiency anemia in the setting of Plummer Vinson syndrome. A typical high risk patient is usually male, >40 years of age, who has a history of a tobacco and alcohol use. High risk sites include the lower lip, lateral or ventral tongue, floor of the mouth, and soft palate.

CLINICAL SIGNS AND SYMPTOMS

- Erythroplakia or leukoplakia
- A lump or thickening in mucosa, gland, or lymph node area
- An ulcer that does not heal

- Abnormal bleeding
 - Pain or numbness
 - Persistent cough or hoarseness
 - Persistent indigestion or dysphagia
 - Histological presentation:
 - Well differentiated
 - Moderately differentiated
 - Poorly differentiated
- By site:
- Lip:
 - 90% of lip carcinomas occur on lower lip
 - Predominantly middle aged males
 - Loss of vermillion architecture
 - Pipe smokers are at increased risk
 - Metastasis is late in the course of the disease
 - 90% 5 year survival
 - Tongue (see Fig. 10–19):
 - Approximately 50% of all intraoral cancer
 - Lateral and ventral surfaces are common
 - Dorsum is rare



FIGURE 10–19. Squamous cell carcinoma. (Also see color insert)

Enlarged red and white lesion of the right lateral tongue growing into the spaces between teeth. The lesion clinically was “rock hard” to palpation. (Courtesy of Dr. John Kacher, Oral Pathology Laboratory, Inc., Flushing, NY.)

- 75% male
- Tobacco and alcohol abuse associated
- Posterior location imparts a poor prognosis
- Floor of mouth:
 - Approximately 30% of all intraoral cancers
 - 90% males
 - Tobacco and alcohol abuse associated
 - 67% 5 year survival if localized; 20% 5 year survival if metastasis to cervical lymph nodes has occurred
- Gingival:
 - Less than 10% of all intraoral cancers
 - Male > female
 - Mandible > maxilla
 - Early bone invasion
 - 33–50% show lymph node metastasis on initial presentation
 - 40% 5 year survival if localized; 10% 5 year survival if metastatic
- Palatal:
 - Approximately 10% of all intraoral cancers
 - Soft palate lesions are more common than hard palate lesions
 - Salivary gland neoplasms and melanomas also occur in this location

TREATMENT

- Lesion needs to be staged (see TMN clinical staging, below)
- Surgical resection followed with radiation or radiation to reduce size of tumor, followed by resection
- Radiation therapy:
 - Can lead to osteoradionecrosis (ORN)
 - Low risk of ORN if radiation dose <45 Gy or 4500 rads
 - High risk of ORN if radiation dose >65 Gy or 6500 rads
- Chemotherapy:
 - Antimetabolites
 - Alkylating agents
 - Antibiotics
 - Vinca alkaloids
 - Can cause:
 - bone marrow suppression leading to thrombocytopenia and agranulocytosis.
 - painful ulcerations.
 - opportunistic infections (e.g., candidiasis, herpes).

PROGNOSIS

- 70% 5 year survival if localized
- 15% 5 year survival if metastasis occurs

TNM Clinical Staging

Grading

T = Tumor	
T0	no evidence of primary tumor
Tis	carcinoma-in-situ
T1	Tumor <2 cm
T2	Tumor >2 cm
T3	Tumor >4 cm
T4	Tumor invasive of deep structures
N = Node	
N0	No nodal involvement
N1	Single ipsilateral node <3 cm
N2a	Single ipsilateral node >3 cm, but <6 cm
N2b	Multiple ipsilateral nodes, <6 cm
N2c	Bilateral or contralateral nodes, <6 cm
N3	Nodes, at least one >6 cm
M = Metastases	
M0	No known metastases
M1	Distant metastasis present

Staging

Stage 0	Tis N0 M0
Stage 1	T1 N0 M0
Stage 2	T2 N0 M0
Stage 3	T3 N0 M0 and T1, T2, or T3 with N1 M0
Stage 4	T4 with any N1 M0 Any T with N2 M0, or N3 M0 Any T, any N, with M1

Verrucous Carcinoma

Chewing and other forms of smokeless tobacco have been associated with verrucous carcinoma. Verrucous carcinomas rarely metastasize, but can be associated with a conventional squamous cell carcinoma. Ten to fifteen percent of patients with verrucous carcinoma will develop a squamous cell carcinoma.

CLINICAL SIGNS AND SYMPTOMS

Appear as large, broad-based, exophytic papillary leukoplakic lesions.

TREATMENT

Complete surgical excision. Radiation is contraindicated.

Proliferative Verrucous Leukoplakia

This is a clinical term used to describe a progressive form of leukoplakia that responds poorly to treatment. It differs from other leukoplakias in several ways:

- Strong female predilection 4:1.
- Tends to spread slowly and involve multiple sites, starting out flat and then becoming exophytic.
- Histologically, the lesions go through stages ranging from hyperkeratosis, dysplasia, verrucous hyperplasia, verrucous carcinoma and eventually to squamous cell carcinoma.
- No effective treatment exists.

Ewing's Sarcoma

A malignant primary tumor of the bone, considered the fourth most common malignancy, likely of neuroectodermal origin. Rarely found in the jaws. Typically found in white males aged 5–25.

CLINICAL SIGNS AND SYMPTOMS

- Intermittent pain
- Swelling
- Fever
- Anemia
- Leukocytosis
- Radiolucent lesion with onion skinning

TREATMENT

- Resection, radiation, and aggressive chemotherapy.

Osteosarcoma

This is an osteoid producing neoplasm from bone and considered to be the second most common malignant neoplasm of the bone.



Ewing's sarcoma—onion skinning.



Osteosarcoma—sunburst appearance.

CLINICAL SIGNS AND SYMPTOMS

- Painful swelling
- Loose teeth
- Paresthesia
- Nasal obstruction
- Epistaxis
- Pathologic fractures
- Ulcerations over the lesion
- Appears as a mixed radiolucent/radiopaque lesion, with aggressive expansion
- Sunburst appearance, symmetrical widening of the PDL
- Elevated alkaline phosphatase

TREATMENT

- Radical resection
- Chemotherapy and/or radiation

Kaposi's Sarcoma

This is a malignant soft tissue tumor typically associated with HIV infection in the setting of AIDS. It is believed to be vascular in origin. Its exact etiology is unknown, but human herpes virus 8 has been recently implicated to have a causative role. Other forms of Kaposi's sarcoma include transplantation associated, lymphadenopathic and chronic KS, often seen in middle aged males from central equatorial Africa and other areas including Russia, Poland, and Italy.

CLINICAL SIGNS AND SYMPTOMS

- Can occur as patches, plaques, or nodules.
- Reddish-purple, thin, oval plaques on skin and mucosa.
- Usually affects skin of lower extremities.
- Oral lesions are common in immunocompromised patients or those with AIDS.
- Oral lesions are often multifocal.

TREATMENT

Antiretrovirals for HIV chemotherapy for lesions; radiation; intralesional vinblastine; liquid nitrogen cryotherapy.

► ODONTOGENIC TUMORS

Ameloblastoma

Ameloblastoma is the most common odontogenic tumor. It arises from odontogenic epithelium. Sources include the developing enamel organ, the epithelial lining of an odontogenic cyst, or the basal cell layer of the oral mucosa.

CLINICAL SIGNS AND SYMPTOMS

Ameloblastoma is a locally aggressive neoplasm, which may reach large sizes, cause bony expansion, and possible expansion and erosion of the cortical plate. The most common form is the *conventional solid* or *multicystic type*, accounting for approximately 86% of all cases. It is found most commonly in the molar ramus area of the mandible and may extend to the symphyseal region. Growth of the tumor tends to be slow and the expansile area may be firm or fluctuant depending on the destruction of the underlying bone. Radiographically, the tumor presents with a unilocular or multilocular lucency with well-defined hyperostotic borders. Variants of ameloblastoma include:

- *Unicystic and plexiform unicystic*: Generally found in younger individuals (teens and young adults), and present in a dentigerous cyst-like manner. Represents approximately 13% of all cases.
- *Peripheral*: Less than 1% of ameloblastomas—present as painless gingival nodules, with no intrabony component.
- *Ameloblastic carcinoma*: Occurs after several unsuccessful attempts at resection of a benign ameloblastoma.
- *Malignant ameloblastoma*: Benign in a distant location; lung is most common metastatic site.

TREATMENT

Patients with conventional solid ameloblastomas are treated with simple enucleation or en bloc resection depending on size and extent of the tumor. The unicystic type can be treated by enucleation, with possible local resection to prevent recurrences. Peripheral ameloblastomas are treated with simple surgical excision. Malignant ameloblastoma and ameloblastic carcinoma have a poor prognosis. Radiotherapy is generally not effective.

Odontogenic Myxoma

This is the most common odontogenic tumor of mesenchymal origin (i.e., precursor cells to the dental follicle).

CLINICAL SIGNS AND SYMPTOMS

- Predilection for posterior mandible
- Honeycombed or multilocular radiographic appearance
- Lesions can cross the midline

TREATMENT

Wide local excision.

Odontogenic Fibroma

Also known as a central odontogenic fibroma. This is another odontogenic tumor of mesenchymal origin. It is an uncommon lesion.

CLINICAL SIGNS AND SYMPTOMS

- Females > males.
- 60% in maxilla.
- Located in the anterior maxilla (anterior to first molar) and posterior mandible.
- Smaller ones usually completely asymptomatic.
- Larger lesions may be associated with localized bony expansion or teeth loosening.
- Radiographically, small odontogenic fibromas tend to be well-defined, unilocular, radiolucent lesions often associated with the apical area of erupted teeth.
- Larger lesions tend to be multilocular radiolucencies. Radiopaque flecks and sclerotic borders may be seen in some tumors. Root resorption of teeth is common and lesions located between teeth may cause root divergence.

TREATMENT

Surgical excision.

Cementoblastoma

This is a benign neoplasm of functional cementoblasts.

CLINICAL SIGNS AND SYMPTOMS

Radiographically appears as a mixed radiolucent-radiopaque lesion consisting of a calcified mass intimately associated with the root. The root outline is obscured by a radiolucent rim. Clinically presents as a slow growing lesion causing expansion of the bony cortex.

TREATMENT

Surgical excision.



FIGURE 6-10



FIGURE 6-13



FIGURE 6-15



A



B



C

FIGURE 8-5A, B and C



A



B



C



D

FIGURE 8-11A, B, C and D



FIGURE 10-3



FIGURE 10-6



FIGURE 10-4A and B



FIGURE 10-5



FIGURE 10-7



FIGURE 10-8



FIGURE 10-11



FIGURE 10-9



FIGURE 10-12



FIGURE 10-10



FIGURE 10-13



FIGURE 10-14A and B



FIGURE 10-15



FIGURE 10-16



FIGURE 10-17A



FIGURE 10-19

Patient Management, Public Health, and Ethics

David E. Webb, DDS

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The Dentist–Patient Relationship

COMMUNICATION

- Verbal and nonverbal communication serve as the foundation of the dentist–patient relationship.
- Verbal and nonverbal communication should be appropriate for each situation according to a patient’s gender, age, cultural background, previous and current experiences with medical/dental care, and life experiences.
- **Verbal communication** should include:
 - clear, descriptive language which avoids terminology not understood by patients.
 - a confirmation that the patient understands what has been discussed (having the patient repeat what has been discussed is often the most effective way).
 - a variety of questions types.

Question Type	Description	Example
Open-ended	Elicits an unguided response. Open-ended questions are the most effective type of question	“How are you today?”
Direct	Elicits specific information	“What about brushing your teeth don’t you like?”
Probing	Elicits additional information that the patient doesn’t originally present	“What else don’t you like about brushing your teeth?”
Leading	Elicits a specific guided response	“How many times a day do you brush your teeth?”
Laundry list	Elicits the selection of one choice from many	“Do you brush your teeth after each meal, daily, or occasionally?”
Facilitating	Elicits patient elaboration without restriction of topic	“How have you been?”



Eyes and eyebrows serve as great indicators for a patient’s pain level.

- **Nonverbal communication** components include:
 - gaze (**eye contact** is the principle nonverbal component)
 - facial expressions
 - gestures
 - posturing
 - touch

ANXIETY AND PAIN CONTROL

Aversive conditions stimulate the following psycho-physiological reactions:

- Fear—results from anticipation of a threat arising from an **external origin**.
 - Dental fears can be evaluated by observing what a patient says and how she/he looks and behaves.
- Anxiety results from anticipation of a threat arising from an **unknown or unrecognized origin**.
 - Anxious patients, resulting from prior traumatic experiences in health care settings, are the **most difficult patients** as they often cause the dentist to become anxious as well.
- Stress results from exposure to an aversive factor which leads to generalized maladaptation of the psycho-physiologic system.

The combination of fear, anxiety, stress, and pain result in behavior modification. Behavior modification can either enhance or lessen the perceived threat.

Nonaversive conditions create a nonthreatening environment. Nonaversive conditions include:

- Data collection
- Problem identification
- Tell-show-do technique
- Informing patient regarding the possibility of pain
- Sharing control with patient—have patient raise hand to stop procedure
- Preventative oral health behaviors

BEHAVIOR MANAGEMENT

The goal of behavior modification is to substitute new behavior in the place of improper behavior. Dentists play a key role in assisting patients to unlearn improper behaviors.

► PUBLIC HEALTH

Fundamental Principles

The most critical aspects of C.E.A. Winslow's definition of public health deal with **prevention** of disease and **promotion** of health through an organized **community effort**.

Disease Prevention Principles

1. A problem exists with potential or actual widespread morbidity/mortality.
2. A solution to the problem exists.
3. A solution to the problem is applied to a **community** (not just an individual).
4. Outcomes assessment ensures efficient problem resolution.

Preventative services implement these principles by providing screenings (i.e., oral cancer), preventative services (i.e., sealants), referrals, and follow up.

The key to health promotion is **education**. Successful education requires active participation (the more involved the children are in school-based programs, the more successful the program will be).



It is more ethical and cost effective to prevent disease rather than treat it.

Epidemiology

Definitions and Terminology

Frequency	Number of individuals having a disease at a given time (#cases)
Prevalence	Proportion or percentage of individuals having a disease at a given time (#cases/population)
Incidence	Rate of new cases of disease per time (#cases/population/time, i.e., the change in prevalence)
Morbidity	Incidence of a specific disease
Mortality	Rate of deaths resulting from a specific disease (#deaths secondary to disease of interest/# total deaths)
Natality	Ratio of births to total population (birth rate)
Birth-death ratio	#Births/#deaths per year. It reflects a population's change in size
Randomized control study	A study which randomly assigns subjects to either a control treatment or test treatment group and follows the groups through time. A study is "blind" if subjects are unaware which group they are assigned and is "double blind" if both examiners and subjects are unaware. Randomization and "blinding" are methods which reduce bias.
False positive	Individual doesn't have the disease but the test reports he/she does
True positive	Individual has the disease which the test confirms
False negative	Individual has the disease but the test reports he/she doesn't
True negative	Individual doesn't have the disease which the test confirms
Sensitivity	Proportion of truly diseased persons who are identified by a screening test as being diseased (measures "how good" a test is at correctly identifying truly diseased persons).
Specificity	Proportion of truly nondiseased persons who are so identified by a screening test (measures "how good" a test is at correctly identifying nondiseased persons).

Dental Epidemiologic Indices

Reversible Indices (Quantify Reversible Conditions)	
Simplified oral hygiene index	Quantifies the amount of debris and/or calculus found on the six surfaces of preselected teeth.
Plaque index (of Silness and Loe)	Approximates plaque thickness at all four gingival margin surfaces of each tooth.
Gingival index (of Loe and Silness)	Assesses the color, consistency, and bleeding on probing of gingiva to quantify the severity of gingivitis.
Sulcus bleeding index	Similar to gingival index except increased emphasis on whether or not gingiva bleeds on probing.
Papillary, marginal, and attached gingival (PMA) Index	Assesses the presence or absence of gingivitis in areas of the papillary, marginal, and attached gingiva.
Periodontal index	Quantifies the status of both gingiva and alveolar bone for each tooth, but places greater emphasis on alveolar bone resorption.
Irreversible Indices (Quantify Irreversible Conditions)	
Decayed, missing, or filled teeth (DMFT) index	Quantifies how the permanent dentition has been affected by dental caries.
Decayed, missing, or filled surfaces (DMFS) index	A more detailed index than DMFT as it quantifies how each tooth surface has been affected by caries.
Decayed, extracted, or filled teeth (deft) index	Quantifies how the primary dentition has been affected by dental caries.
Decayed, extracted, or filled surfaces (defs) index	Quantifies how each surface of the primary dentition has been affected by dental caries.

SPin/SNout- positive results from SPecific tests rule *in* disease, while negative results from SeNsitive tests rule *out* disease.

Managed Care and Quality Assurance

Managed care occurs when a third-party payer mediates between providers and their patients. The goal of managed care organizations (MCOs) is to **control health care costs** while **maintaining the quality of care**.

- Controlling health care costs:
 - MCOs control costs by placing limits on provider's fees and providing treatment guidelines.

Preferred Provider Organization—PPOs prefer their members to use contracted providers, but still reimburse care rendered from noncontracted providers.

- Managing quality of care:
 - MCOs constantly assess the quality of care provided (quality assessment) as well as implement beneficial changes (quality assurance).
 - Utilization reviews consider how services are both requested and performed and allow MCOs to establish standards of care.
- Two types of managed care are:
 - Health maintenance organizations (HMOs):
 - Third-party payers who contract a network of providers to provide discounted services for their members.
 - Providers contract with HMOs in an effort to ensure more patients and in return agree to discount their services.
 - Providers render care following a set of care guidelines provided by the HMO.
 - Preferred provider organizations (PPOs):
 - Like HMOs, health care providers contract with PPOs to ensure large patient pools and are obligated to provide health care at reduced rates for PPO members.
 - Unlike HMOs, PPOs will reimburse members who receive care by a non-PPO contracted provider albeit at a reduced rate.
- Providers are reimbursed by third-party payers in a variety of methods:
 - Capitation:
 - Provider receives a set amount for each patient each month regardless of care rendered.
 - Usual, customary, and reasonable (UCR) fee:
 - Consensus of the usual fee providers charge in a geographic area for a specific service.
 - Fee schedule (table of allowance):
 - Patients pay the difference between the provider's fee and the amount the insurance company covers.

Infection Control

PATHOGEN TRANSMISSION AND RESULTANT DISEASES

Transmission of infection requires:

1. Source of infectious microorganisms
2. Susceptible host
3. Means of transmission for the microorganism

SIX ROUTES OF TRANSMISSION

1. Droplet transmission—droplets containing microorganisms, generated when infected persons cough, sneeze, or talk, contact surfaces of the eye, nose, or mouth.
2. Airborne transmission—droplet nuclei (residue from evaporated droplets) or dust particles containing microorganisms enter the upper and lower respiratory tracts. These nuclei can remain suspended in air for long periods of time.
3. Vector-borne—an insect or other animal transmits the pathogen from one host to another.
4. Indirect (fomite)—susceptible person is infected from contact with a contaminated surface.

DAVID Failed the pathogen transmission quiz.
Droplet
Airborne
Vector-borne
Indirect
Direct
Fecal-Oral

5. Direct—requires physical contact between an infected and a susceptible person, and the physical transfer of microorganisms. Includes vertical transmission (transmission that occurs in utero, during childbirth, and via breast milk) and reproductive transmission.
6. Fecal-oral—microorganisms enter the body after ingesting contaminated food and/or water. They multiply inside the digestive system and are eventually shed in feces. Feces may then contaminate a water supply if inadequate sewage treatment and water filtration measures are not in place.

PATHOGENS

As a result of the human immunodeficiency virus (HIV) epidemic, the Center for Disease Control instigated a Standard (Universal) Precaution in 1985 with the goal to prevent occupational transmission of infectious diseases while providing care. It targets the most infectious agent (Hepatitis B) thus precluding other cross-infections (see Table 11–1).

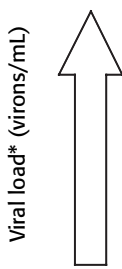
HEPATITIS B VIRUS (HBV)

- The **greatest blood-borne risk** for health care workers.
- Most infectious blood-borne pathogen known and is transmitted by percutaneous or mucosal exposure to blood or body fluids of an infected person and results in acute and chronic liver infections.

HEPATITIS C VIRUS (HCV)

- Acute and chronic hepatitis may also result from HCV infections transmittable via blood; thus high risk groups include IV drug users, individuals with tattoos or body piercings, transfusion recipients, and those with blood dyscrasias requiring clotting factors.

Table 11–1. Pathogens and Risk of Infection

	Source	Average risk for infection after needle stick or cut exposure with viral-infected blood
	HBV	6–30% (for susceptible persons)
	HBsAg+, HBeAg+	22.0–31.0% clinical hepatitis; 37–62% serological evidence of HBV infection
	HBsAg+, HBeAg–	1.0–6.0% clinical hepatitis; 23–37% serological evidence of HBV infection
	HCV	1.8%
	HIV	0.3%

*Increased viral load correlates with increased postexposure infection.

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

- Retrovirus which is transmitted by sexual contact or sharing needles with an infected person, and through transfusions of infected blood or blood clotting factors (rare now as blood is screened for HIV antibodies). Babies born to HIV-infected women may become infected in utero, during birth, or through breast-feeding.

MYCOBACTERIUM TUBERCULOSIS

- Bacterium responsible for tuberculosis is carried in airborne droplet nuclei generated when infected persons sneeze, cough, or speak.
- Particles cause infection when susceptible individuals inhale them.
- The immune response usually limits the spread of *M. tuberculosis* to the lungs, where latent tuberculosis (TB) may persist for years.
- Persons with latent TB have no symptoms but exhibit reactive tuberculin skin tests (TST) and require treatment to prevent active TB infections. Only active, not latent, TB poses a risk for transmission.

Barriers

Standard precautions require gown and gloves when exposure to blood and/or saliva is anticipated. Providers must ensure employee compliance with these standards.

Protective Attire

Protective Clothing	Should be changed at least daily or when visibly soiled. Long sleeves and high neck are required. Permeability of protective clothing is dictated by procedure (fluid-resistant gowns only required when large fluid exposures are likely).
Gloves	Change between all patients.
Material	Indications/Hypersensitivity Reactions
Natural rubber latex	■ Derived from rubber tree (<i>Hevea brasiliensis</i>) sap. ■ Contains potentially antigenic proteins which can leach out during perspiration and cause immediate hypersensitivity reactions (type I, IgE-mediated). ■ Prior latex exposure can cause Delayed Hypersensitivity reactions (type IV) secondary to chemicals used in glove manufacturing.

(Continued)

	■ Atopic individuals are at increased risk for hypersensitivity reactions, but individuals with seasonal allergies are not.
“Hypoallergenic”	■ Chemical coating over latex may still result in hypersensitivity reactions so not suitable for latex allergic individuals.
Vinyl and Nitrile	■ Suitable for latex allergic individuals. ■ Nitrile or heavy-duty utility gloves recommended when handling hazardous chemicals or cleaning dental office.
Facemask	Change between all patients or during care if inner or outer surface becomes moist. Must be >95% effective at filtering small particle aerosols (1–3 µm).
Protective eyewear	Must have solid side shields. Face shields are acceptable.

Barrier Techniques

Impervious-backed paper, aluminum foil, or plastic covers should be used to protect items and surfaces (e.g., light handles or X-ray unit heads) that may become contaminated by blood or saliva during use and that are difficult or impossible to clean/disinfect. Between patients, the coverings should be removed (while dental care workers are gloved), discarded, and replaced (after ungloving and washing of hands) with clean material.

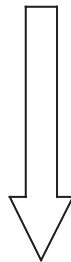
Appropriate use of rubber dams, high-velocity air evacuation, and proper patient positioning should minimize the formation of droplets, spatter, and aerosols during patient treatment. In addition, splash shields should be used in the dental laboratory.



*Hands are the most **common**
mode of pathogen
transmission.*

Hand Hygiene Definitions and Descriptions

Increasing efficacy



Handwashing

Washing hands with plain soap and water. Plain soap physically **reduces bacterial counts**, without exhibiting bacteriostatic (inhibit bacterial growth or multiplication) or bacteriocidal (kill bacteria) effects.

Antiseptic hand wash

Washing hands with water and soap containing an antiseptic agent (i.e., triclosan, chlorhexidine gluconate, iodophors, and parachlorametaxlenol). Triclosan is bacteriostatic, while chlorhexidine gluconate is both bacteriostatic and bacteriocidal.

Alcohol-based hand rub (waterless hand hygiene)

Rubbing hands with an alcohol-containing preparation (i.e., 60–95% ethanol or isopropanol alcohol-containing preparation). Alcohol-based hand rubs are **bacteriocidal**, sporicidal, and fungicidal.

Surgical antisepsis

Antiseptic hand wash or alcohol-based hand rub performed preoperatively to **eliminate** microorganisms on hands. Long-lasting antimicrobial activity required.

Health care workers should wash hands:

- when visibly dirty
- after touching contaminated objects with bare hands
- before glove placement and immediately after glove removal

Latex Allergies

Type I	Immediate hypersensitivity reaction to natural rubber latex proteins. Common presentation: urticaria (hives), rhinorrhea, itchy eyes, burning skin sensation. More severe reaction: dyspnea, anaphylaxis (shock), and death. Results in a more severe and immediate systemic reaction than contact dermatitis.
Type IV	see Allergic Contact Dermatitis

Contact Dermatitis

Irritant	Most common adverse epithelial reaction occurring in 20–30% of health care workers either occasionally or chronically. Develops as dry, itchy, irritated areas on the skin around the contact area. Commonly associated with and aggravated by frequent handwashing, residual powder left on hands, and harsh antiseptic handwashing agents. Not an allergic reaction.
Allergic	Type IV or delayed hypersensitivity reaction due to contact with chemical allergen (i.e., accelerators and other chemicals used in the manufacture of patient-care gloves). Reactions are generally localized to the contact area and occur over a 12–48 hour period.

INSTRUMENT PROCESSING

Instrument processing consists of sequential cleaning, packaging, sterilization, and storage, each occurring at a different area.

Cleaning

Purpose	Cleaning involves the physical removal of debris and reduces the number of microorganisms on instruments. Cleaning is the most important step in sterilization — if visible debris or organic matter is not removed, it can interfere with the sterilization process.
Types	Automated
Ultrasonic cleaner	Removes debris by a process called cavitation (waves of acoustic energy are propagated in aqueous solution to disrupt bonds that hold particulate matter to instruments). Safest and most effective method of cleaning.
Instrument washer	Removes residue on instruments which ensures safe handling for packaging prior to sterilization.

(Continued)

Cleaning (Continued)

Washer-disinfector	Removes residue on instruments and provides low-level disinfection by using a high temperature cycle prior to terminal sterilization.
Manual	Instruments are soaked in a rigid container filled with detergent, disinfectant/detergent, or an enzymatic cleaner before hand washing. Soaking prevents drying of patient material on instruments and makes manual cleaning more efficient.

Sterilization Definitions

Sterilization	Use of a physical or chemical procedure to destroy all forms of life (including bacterial spores).
Disinfection	Use of a chemical procedure to destroy the majority of pathogenic microorganisms, but does not destroy bacterial spores .
Disinfectant	A chemical agent applied to inanimate objects which kills or prevents growth of pathogenic microorganisms (excluding bacterial spores). Pump > aerosol spray and water-based > alcohol-based.
Antiseptic	A chemical agent applied to living tissues which temporarily decreases the concentration of normal flora as well as accumulated transient microorganisms.
Sanitation	Treatment of water to reduce microbial levels to public health-approved levels.
Pasteurization	Heat treatment of dairy food products for short intervals to destroy pathogens (mycobacterium tuberculosis being the primary target).
Critical items	Items that enter sterile body tissues or vascular system (i.e., surgical instruments) that require sterilization.
Semicritical items	Items that contact mucous membranes (dental instruments) that require a high level of disinfection.
Noncritical items	Items that contact intact skin (i.e., dental operator, wheelchairs) require intermediate level of disinfection, whereas items that contact environmental surfaces (i.e., walls, floors) require low levels of disinfection.

Sterilization/Disinfection Overview

Process	Microorganisms Destroyed	Method	Examples	Patient-Care Application
Sterilization	All microorganisms including bacterial spores [†]	Heat automated High temp	Steam (autoclave), dry heat, rapid heat transfer, unsaturated chemical vapor	Heat-tolerant critical and semicritical items
		Low temp	Ethylene oxide gas, plasma sterilization	Heat-sensitive critical and semicritical items
		Liquid immersion	2% Glutaraldehyde (not recommended for surgical instruments)	
High-level disinfection	All microorganisms except high numbers of bacterial spores	Heat automated Liquid immersion	Washer-disinfector 2% Glutaraldehyde, 6% hydrogen peroxide, peracetic acid	Heat-sensitive semicritical items
Intermediate-level disinfection	Mycobacterium tuberculosis*, vegetative bacteria, most viruses, most fungi (not spores)	Liquid contact	Chlorine compounds, alcohols, iodophors, phenolics, quaternary ammonium compounds (all requiring tuberculocidal activity)	Noncritical item with visible blood
Low-level disinfection	Vegetative bacteria, certain fungi and viruses (mycobacterium tuberculosis and spores not destroyed)	Liquid contact	Quaternary ammonium compounds, some phenolics, some iodophors	Noncritical item without visible blood

[†]Bacterial spore destruction (i.e., *Clostridium botulinum*) is regarded as the benchmark for effective sterilization

*Mycobacterium tuberculosis destruction is regarded as the benchmark for effective disinfection

Antimicrobial Method	Mechanism of Action/Notes
Steam (autoclave)	Heat inactivation of critical enzymes and proteins. Applies heat under pressure to increase the temperature above boiling, as 100°C is insufficient to destroy spores. Most efficient sporicide, fungicide, and viricide available. Bacterial spores should be cultured weekly to determine autoclave efficacy.
Dry heat	Heat inactivation of critical enzymes and proteins. Has poor penetrating power so instruments must be dried prior to sterilization and subjected to long cycles, but does not dull or corrode metal instruments.
Rapid heat transfer	Same mechanism as dry heat sterilization, except higher temperatures and controlled air flow (forced air) within the chamber provide shorter cycles.
Unsaturated chemical vapor	Heat inactivation of critical enzymes and proteins similar to steam but uses a solution of alcohol, formaldehyde, ketone, acetone , and water rather than distilled water to produce sterilizing vapor. Instruments must be dried completely before sterilization, but sterilization does not dull or corrode metal instruments because of low level of water present during the cycle.
Ethylene oxide gas	Alkalating agent that irreversibly inactivates DNA and proteins. It is a toxic, flammable, highly penetrative gas used for the sterilization of plastic, rubber and other heat-sensitive items. Instruments must be thoroughly cleaned and dried prior to slow sterilization (10–16 hours) with ethylene gas.
Iodine	Oxidizing agent irreversibly combines with proteins. Most effective skin antiseptic used. Preparations which combine iodine and a solubilizing agent are considered iodophors (i.e., Betadine). Iodophors can penetrate the wax and outer lipid layers of mycobacteria.
2% Glutaraldehyde	Alkalating agent will inactivate all microorganisms with sufficient contact time (10 hours). Most potent chemical germicide, but allergenic and highly toxic to tissues.
Hydrogen peroxide, Peracetic acid	Oxidizing agents of both these peroxides are microcidal. Clinical efficacy and use is determined by preparation strength.
Chlorine compounds	Oxidizes free sulfhydryl groups rendering bacteria and most viruses inactive. Active constituent of bleach (hypochlorite).

(Continued)

Antimicrobial Method	Mechanism of Action/Notes
Phenols	Oxidizing agent. Was the prototype hospital disinfectant but discontinued as deemed too caustic. Able to penetrate the wax and outer lipid layers of mycobacteria.
Quaternary ammonium compounds	Disrupt cell membranes via amphipathic interaction. These cationic detergents are widely used for skin antiseptics and as disinfectants because they are effective against gram-positive bacteria. No sporicidal, tubercidal, or viricidal activity. Inactivated by anionic detergents (soaps) and iron found in hard water.
Anionic surface-acting materials	Anionic detergents and soaps facilitate mechanical removal of microbes by decreasing surface tension on the skin surface.
Alcohols	Denatures proteins , extracts membrane lipids, and acts as a dehydrating agent. Most widely used antiseptic . Effective against lipophilic viruses except when harbored in dried blood, saliva, and other secretions (thus alcohols are not suitable for surface cleansing agents). Alcohol swabs are 70% ethanol, whereas alcohol-based hand rubs are 90–95% isopropyl alcohol.
Filtration	Sterilize liquids by physically restricting the flow of microbes larger than the pore size of the filter. A nitrocellulose filter with a pore size of 0.22 μm restricts all bacteria.

Heat transfer Method	Temperature °F (°C)	Time	Pressure Generated
Steam (autoclave)	250 (121)	15–20 min	15 psi
“Flash cycle”	270 (132)	3 min unwrapped	30 psi
Unsaturated chemical vapor	270 (132)	20–40 min	20 psi
Dry heat	320 (160) 340 (171)	2 hrs 1 hr	NA
Rapid heat transfer	375 (191)	6 min unwrapped, 12 min wrapped	



Heat sterilization is the most efficient and monitorable method of sterilization possible.



250°F (121°C) is the minimum temperature required to annihilate all microbes.

Waste Disposal

Waste Category	Definition/Examples	Disposal Method
Unregulated waste	Items that may have had contact with blood, or secretions but do not pose substantial risk of causing infection (used gloves, masks, gowns, lightly soiled cotton rolls, or gauze)	Dispose with ordinary waste
Regulated waste	Infectious waste that carries a substantial risk of causing infection during handling and disposal	
Sharps	Needles, scalpel blades, orthodontic bands, burs	Sharps container (must be puncture resistant, color coded, and leak proof)
Nonsharp solids	Blood-soaked gauze, extracted teeth, items caked with dried blood, or other potentially infectious material	Biohazard bag (a color-coded or labeled container that prevents leakage)
Liquids	Blood, suctioned fluids	Pour waste carefully into a drain connected to a sanitary sewer system*

*State and local regulations dictate what volume of blood or other body fluids can be discharged into the sanitary sewer and if pretreatment or neutralization is required.

Occupational Exposure

Dental health care workers are at risk for acquiring influenza, measles, mumps, rubella, varicella, tuberculosis, hepatitis B and C, and HIV. All of these diseases, excepting hepatitis C and HIV, are vaccine-preventable.

When an exposure does occur, the site should be immediately washed with soap and water (mucous membranes should be flushed with water) and evaluated by a qualified health care professional who will provide counseling, postexposure management, and follow up. An exposure report must be generated according to federal and state requirements.

Exposure risk may be reduced by implementing:

1. Technologically based engineering controls (self-sheathing anesthetic needles, retractable scalpels)
2. Behavior-based work practice controls (“scoop” technique to recap anesthetic needles, restricting the use of fingers during suturing and when administering anesthesia)
3. Appropriate personal protective equipment (gloves, masks, protective eye-wear with side shields, and gowns)

Operatory Equipment Infection Control

- Noncritical items:
 - Use surface barriers to protect clinical contact surfaces (i.e., light switches, chair controls) when possible.
 - Disinfect surfaces when surface barriers are not possible.
- Operatory waterlines:
 - Must meet drinking water standards.
 - Any waterline used during patient care must be discharged for at least 20 seconds after each patient.



*Do not disinfect any
equipment that can be
sterilized!*

► ETHICS & LEGAL ISSUES

Ethical Principles

BENEFACTANCE

- “Do good.”
- Dentists are to render competent and timely care in an effort to benefit the patient.

PATIENT AUTONOMY

- “Self-governance.”
- Dentists are to treat patients according to the patient’s desires and protect patients’ confidentiality.

NONMALEFACTANCE

- “Do no harm.”
- Dentists are to keep skills and knowledge up-to-date and practice within their limits in order to protect the patient from harm.

JUSTICE

- “Fairness.”
- Dentists are to deal justly with patients, colleagues, and society.

VERACITY

- “Truthfulness.”
- Dentists are to be honest and trustworthy, communicating truthfully and without deception.

BAN the **JV** team for
being unethical!
Beneficence
Autonomy (patient)
Nonmaleficence
Justice
Veracity



Child abuse most commonly occurs in children below the age of three.



The majority of domestic violence involves facial injuries.

JURISPRUDENCE

The theory and philosophy of law.

GOOD SAMARITAN LAW

- Ensures that health care providers are protected from lawsuits while rendering emergent care in accidents.
 - Provided that care rendered does not demonstrate gross negligence.
- Dentists are not included in this law in all states.

Dentists are legally obligated to immediately notify designated state agencies upon observation of **domestic violence** or suspected **abuse** injuries.

AMERICANS WITH DISABILITIES ACT

- Guarantees equal opportunity for individuals with disabilities in public accommodations (dental offices), employment (dental employees), transportation, state and local government services, and telecommunications.
- Defines disability as, “a physical or mental impairment that substantially limits one or more major life activities, has a record of such an impairment, or is regarded as having such an impairment.”
 - Persons with HIV or AIDS are protected under the Americans with Disability Act.

Some implications of this law are that dentists cannot deny care secondary to a disability, dental offices must be structurally accessible to individuals with disabilities, and dentists cannot dismiss or refuse to hire employees because of a disability.

INFORMED CONSENT

The elements of informed consent are:

- **Who?** Who will render the treatment?
- **What?** What are the treatment alternatives (including no treatment) and what treatment will be done?
- **When?** When will the treatment occur? (i.e., a temporary crown will be placed today and a definitive crown will be delivered in one week)
- **Where?** Where will the treatment occur? (in cases requiring referral)
- **Why?** Why will the treatment be rendered? (risks and benefits of all treatment alternatives need to be understood)
- **How?** How much is the fee?

Case-Based Component

David Tavelin, DDS, MD

Case 1	345
Case 2	351
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CASE 1

Patient Summary

Sex: F
Age: 60
Ht: 5'5"
Wt: 130 lb (59 kg)

Vital Signs

BP: 138/86 mm Hg
Pulse: 72
Resp. Rate: 13

Chief Complaint

"My gums bleed when I brush my teeth."

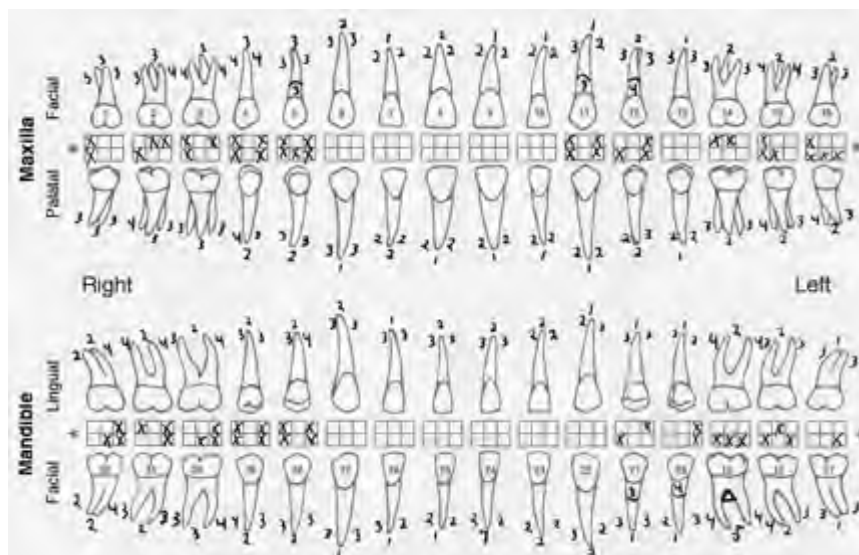
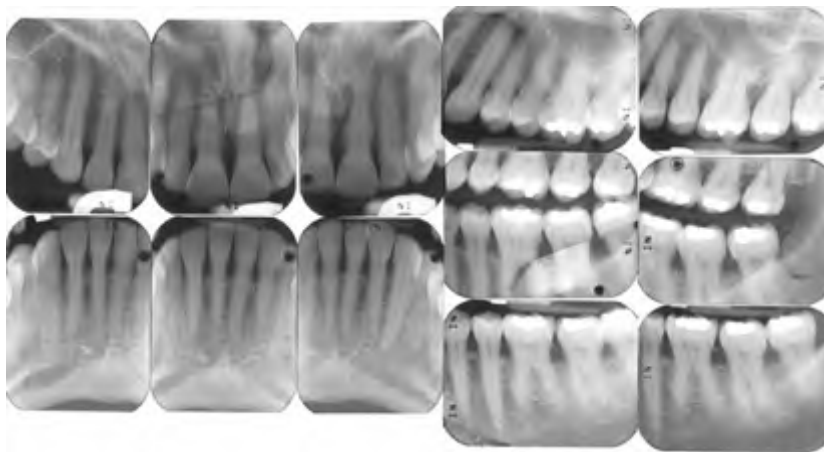
Medical History:

- | | Yes | No |
|---|-------------------------------------|-------------------------------------|
| 1. Are you in good health? | ☒ | ☐ |
| 2. Has there been a change in your health the last year? | ☐ | ☒ |
| 3. Are you under the care of a physician? | ☒ | ☐ |
| 4. Have you been hospitalized in the past 10 years? | ☐ | ☒ |
| Reason for hospitalization: _____. | | |
| 5. Do you have or have you had any of the following conditions: | | |
| Damaged heart valves | ☐ | ☒ |
| Artificial heart valves | ☐ | ☒ |
| Heart murmur | ☐ | ☒ |
| Rheumatic heart disease | ☐ | ☒ |
| Heart attack | ☐ | ☒ |
| Angina | ☐ | ☒ |
| High blood pressure | ☒ | ☐ |
| Stroke | ☐ | ☒ |
| Asthma | ☐ | ☒ |
| Seizures | ☐ | ☒ |
| Fainting spells | ☐ | ☒ |
| Diabetes | ☒ | ☐ |
| Hepatitis/jaundice/liver disease | ☐ | ☒ |
| AIDS/HIV infection | ☐ | ☒ |
| Emphysema/bronchitis | ☐ | ☒ |

Arthritis	☐	☒
Kidney disease	☐	☒
Tuberculosis	☐	☒
Low blood pressure	☐	☒
Sexually transmitted disease	☐	☒
Epilepsy	☐	☒
Cancer	☐	☒
Blood disorder/anemia	☐	☒
6. Are you taking any of the following types of medications?		
Antibiotics	☐	☒
Aspirin	☐	☒
Insulin	☒	☐
Nitroglycerin	☐	☒
Anticoagulants	☐	☒
Antihypertensives	☒	☐
Steroids	☐	☒
Oral contraceptives	☐	☒
Other, please name _____		
7. Do you have an allergy to any of the following?		
Local anesthetics	☐	☒
Penicillin	☐	☒
Sulfa drugs	☐	☒
Barbiturates/sedatives	☐	☒
Aspirin	☐	☒
Narcotics/codeine	☐	☒
Rubber goods/latex	☐	☒
8. Do you smoke or use any tobacco products?	☐	☒
9. Are you pregnant?	☐	☒
10. Do you wear any dental appliances?	☐	☒
11. Have you had trouble associated with previous dental treatment?	☐	☒
If so, please describe _____.		

Dental History

Last dental visit 6 years ago.



1. This patient's blood pressure reading indicates inclusion into which blood pressure classification?
 - a. normal blood pressure
 - b. high normal blood pressure
 - c. stage I hypertension
 - d. stage II hypertension
 - e. stage III hypertension
2. Upon further questioning, the patient informs the dentist that she is currently taking Norvasc (amlodipine). This medication is part of which drug group?
 - a. diuretic
 - b. β -blocker
 - c. α -blocker
 - d. calcium channel blocker
 - e. ACE inhibitor
3. Before treating any insulin-dependent diabetic patient, which of the protocol should be followed in the effort to avoid any episode of insulin shock?
 - a. advise the patient to take the normal dose of insulin prior to visit.
 - b. advise the patient to eat meals as they normally would prior to visit.
 - c. schedule a morning appointment.
 - d. maintain a supply of quick sugar in the event of insulin reaction.
 - e. all of the above.
4. The angulation error in the mandibular anterior periapical views can be corrected by:
 - a. increasing the vertical angulation
 - b. decreasing the vertical angulation
 - c. increasing the horizontal angulation
 - e. decreasing the horizontal angulation
5. This patient's periodontal diagnosis is:
 - a. generalized mild-to-moderate chronic periodontitis
 - b. generalized moderate chronic periodontitis with localized severe periodontitis
 - c. gingivitis
 - d. generalized moderate-to-severe chronic periodontitis with localized mild periodontitis
 - e. localized severe chronic periodontitis
6. The patient exhibits gingival recession on teeth #5, #11, #12, #20, #21 and complains of sensitivity with hot and cold in those areas. These signs and symptoms suggest:
 - a. localized severe acute periodontitis
 - b. attrition
 - c. toothbrush abrasion
 - d. abfraction

7. The facial gingiva of tooth #24 has a measurement of 5 mm of keratinized tissue. This scenario illustrates a/an:
 - a. fenestration
 - b. pseudopocket
 - c. mucogingival defect
 - d. attrition
 - e. all of the above
8. The dentist properly detected furcal bone loss on tooth #19 with:
 - a. a Williams probe from the lingual approach
 - b. an explorer from the lingual approach
 - c. a Williams probe from the buccal approach
 - d. an explorer from the buccal approach

1. **The answer is B.** Memorize the table below:

Classification	Systolic (mm Hg)		Diastolic (mm Hg)
Optimal	<120	and	<80
Normal	<130	and	<85
High normal	130–139	or	85–89
Stage I hypertension (mild)	140–159	or	90–99
Stage II hypertension (moderate)	160–179	or	100–109
Stage III hypertension (severe)	≥180	or	≥110

2. **The answer is D.** Calcium channel blockers affect calcium entry into the heart and vasculature. Blood vessels are relaxed, increasing flow, and reducing workload.
3. **The answer is E.** Review all preventative and emergency protocols.
4. **The answer is B.** The angulation error is foreshortening. When the inferior border of the mandible is delineated in the mandibular anterior view, the image is foreshortened.
5. **The answer is B.** Periodontitis is considered generalized when at least 30% of oral sites contain that degree of bone loss. Severity depends upon degree of clinical attachment loss. Mild is 1–2 mm, moderate is 3–4 mm, and severe is ≥5 mm.
6. **The answer is C.** Toothbrush abrasion is classically seen on the cuspids and bicuspid. Patients brush too hard and eventually suffer from sensitivity.
7. **The answer is C.** A mucogingival defect occurs when loss of attachment advances into the nonkeratinized mucosa. If there is only 5 mm of gingiva, and the pocket depth of #24 buccal is 7 mm, we know that the pocket has advanced past the mucogingival junction.
8. **The answer is C.** The furcation defect was on the buccal and detected with a Williams periodontal probe, not an explorer.

CASE 2

Patient Summary

Sex: F
Age: 69
Ht: 5'3"
Wt: 127 lb.(58 kg)

Vital Signs

BP: 126/78
Pulse: 68
Resp. Rate: 12

Chief Complaint

"I lost my dentures and I want new ones."

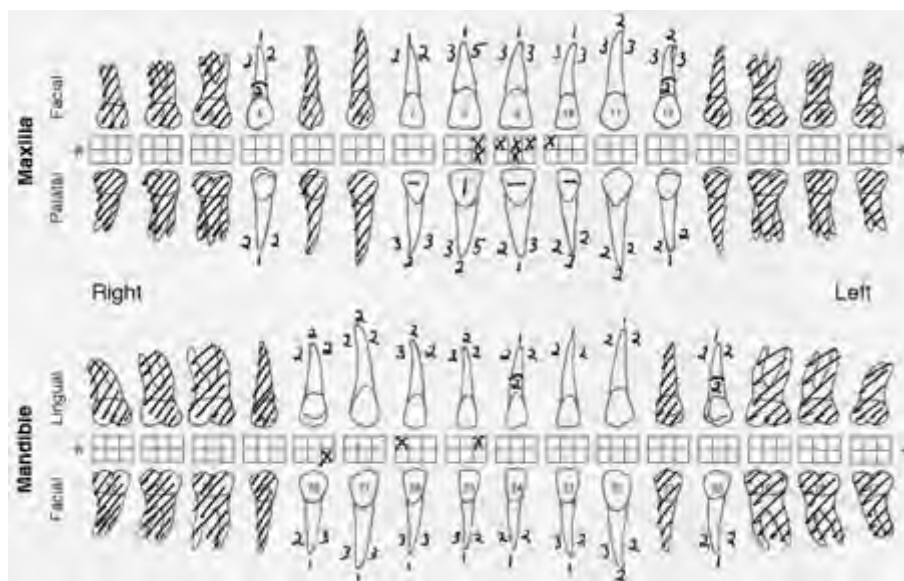
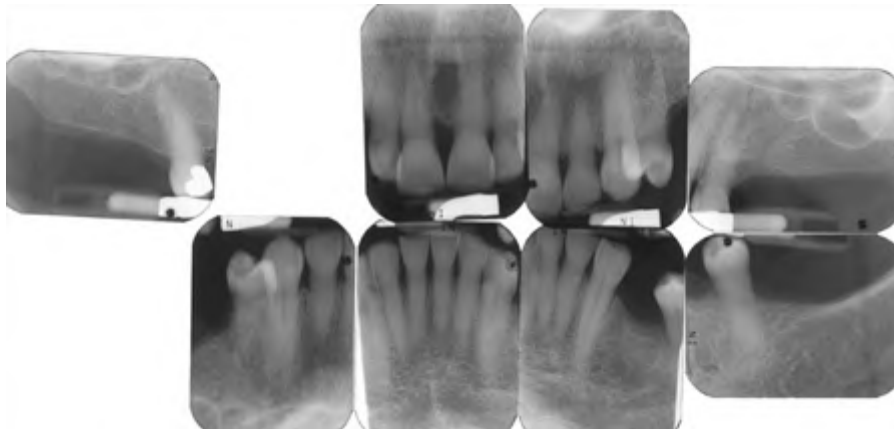
Medical History

	Yes	No
1. Are you in good health?	☒	☐
2. Has there been a change in your health the last year?	☐	☒
3. Are you under the care of a physician?	☒	☐
4. Have you been hospitalized in the past 10 years?	☒	☐
Reason for hospitalization: <u>I had a stroke 2 years ago.</u>		
5. Do you have or have you had any of the following conditions:		
Damaged heart valves	☐	☒
Artificial heart valves	☐	☒
Heart murmur	☒	☐
Rheumatic heart disease	☐	☒
Heart attack	☐	☒
Angina	☐	☒
High blood pressure	☐	☒
Stroke	☒	☐
Asthma	☐	☒
Seizures	☐	☒
Fainting spells	☐	☒
Diabetes	☐	☒
Hepatitis/jaundice/liver disease	☐	☒
AIDS/HIV infection	☐	☒
Emphysema/bronchitis	☐	☒
Arthritis	☐	☒

Kidney disease	☐	☒
Tuberculosis	☐	☒
Low blood pressure	☐	☒
Sexually transmitted disease	☐	☒
Epilepsy	☐	☒
Cancer	☐	☒
Blood disorder/anemia	☐	☒
6. Are you taking any of the following types of medications?		
Antibiotics	☐	☒
Aspirin	☐	☒
Insulin	☐	☒
Nitroglycerin	☐	☒
Anticoagulants	☒	☐
Antihypertensives	☐	☒
Steroids	☐	☒
Oral contraceptives	☐	☒
Other, please name <u>Coumadin (warfarin)</u>		
7. Do you have an allergy to any of the following?		
Local anesthetics	☐	☒
Penicillin	☐	☒
Sulfa drugs	☐	☒
Barbiturates/sedatives	☐	☒
Aspirin	☐	☒
Narcotics/codeine	☐	☒
Rubber goods/latex	☒	☐
8. Do you smoke or use any tobacco products?	☒	☐
9. Are you pregnant?	☐	☒
10. Do you wear any dental appliances?	☒	☐
11. Have you had trouble associated with previous dental treatment?	☐	☒
If so, please describe _____.		

Dental History

Last dental visit 1 year ago.



1. The dentist orders a medical consult and determines that the patient's heart murmur is functional. Antibiotic prophylaxis for subsequent scaling visits should include:
 - a. 2 g penicillin VK 1 hour prior to visit
 - b. 2 g amoxicillin 1 hour prior to visit
 - c. 600 mg clindamycin 1 hour prior to visit
 - d. 500 mg azithromycin 1 hour prior to visit
 - e. no antibiotic prophylaxis necessary
2. The treatment plan includes extraction of #28. The patient's INR just prior to the procedure is 2.0. The dentist should:
 - a. conclude that it is safe to proceed with the extraction
 - b. not perform the extraction at that time
 - c. reschedule the patient and instruct the patient to discontinue coumadin therapy 24 hours before the extraction and begin again 48 hours after
 - d. wait for the INR to rise before beginning the extraction
3. Amoxicillin is:
 - a. bacteriostatic and inhibits bacterial cell metabolism
 - b. bacteriocidal and inhibits 30S ribosomal unit
 - c. bacteriocidal and inhibits cell wall synthesis
 - d. bacteriostatic and inhibits cell wall synthesis
 - e. bacteriocidal and inhibits 50S ribosomal unit
4. The dentist may not use local anesthetic with epinephrine to treat this patient.
 - a. True
 - b. False
5. Reactions to latex might include:
 - a. irritant contact dermatitis
 - b. allergic contact dermatitis
 - c. acute allergic reaction
 - d. all the above
6. This patient's mandibular Kennedy classification is:
 - a. Class II, Modification I
 - b. Class I Modification I
 - c. Class I
 - d. Class III, Modification II
 - e. Class II, Modification II
7. The radiolucent area lying between the roots of teeth #8 and #9 is most likely
 - a. the incisive canal
 - b. the incisive foramen
 - c. representative of periapical pathology
 - d. a nasopalatine duct cyst

8. Blunting of the incisal edges of the mandibular central and lateral incisors is a result of:
 - a. abfraction
 - b. abrasion
 - c. attrition
 - d. caries
 - e. none of the above
9. In constructing the new mandibular RPD, the dentist decides to place indirect retention. Where should it be placed?
 - a. teeth #20 and #28
 - b. teeth # 22 and #27
 - c. tooth #22
 - d. tooth #27
 - e. none of the above
10. Smoking may have all the following effects on the oral cavity, except:
 - a. increased horizontal bone loss
 - b. impaired healing
 - c. halitosis
 - d. increased bleeding on probing
 - e. none of the above (i.e., they are all exhibited in a smoker's oral cavity.)

1. **The answer is E.** Review the American Heart Association Guidelines of antibiotic prophylaxis for bacterial endocarditis.
2. **The answer is A.** A patient on anticoagulants should be managed carefully at times of invasive procedure. Routine stoppage of anticoagulant therapy before extractions is currently a hotly debated issue. It is generally accepted that an INR of less than 4.0 is acceptable for a simple dental extraction.
3. **The answer is C.** Penicillins are bacteriocidal and inhibit cell wall synthesis. Cephalosporins, vancomycin, and bacitracin do the same.
4. **The answer is B.** The use of anesthetic with epinephrine is contraindicated/should be used limitedly in patients with uncontrolled hyperthyroid disease, uncontrolled hypertension, bisulfate allergy, angina, recent myocardial infarction (MI), or who take non-specific beta blockers.
5. **The answer is D.** Irritant contact dermatitis is indicated by erythema, mild edema, and scaling. Allergic contact dermatitis is indicated by actual allergic, delayed-type hypersensitivity type IV reaction. (skin erythema and edema). An acute allergic reaction is type I, IgE mediated, and the most serious, may lead to anaphylactic shock.
6. **The answer is B.** Class I is bilateral distal edentulousness. Class II is unilateral distal edentulousness. Class III is a tooth-bound edentulous space. Class IV is a tooth-bound edentulous area that crosses the midline.
7. **The answer is B.** Review normal radiographic anatomy.
8. **The answer is C.** Abfraction is deformation of cervical enamel due to occlusal forces. Abrasion is wear resulting from external activity like overzealous toothbrushing. Attrition is wear resulting from some natural activity.
9. **The answer is B.** Indirect retention serves to reduce the chance a partial denture is dislodged via lifting in the posterior (as when sticky foods are chewed).
10. **The answer is D.** Smoking causes vasoconstriction in gingival tissues. There would actually be less bleeding on probing in a smoker.

CASE 3

Patient Summary

Sex: M
Age: 45
Ht: 5'10"
Wt: 190 lb. (87 kg)

Vital Signs

BP: 138/84
Pulse: 74
Resp. Rate: 15

Chief Complaint

"I would like to fill the gaps in my teeth."

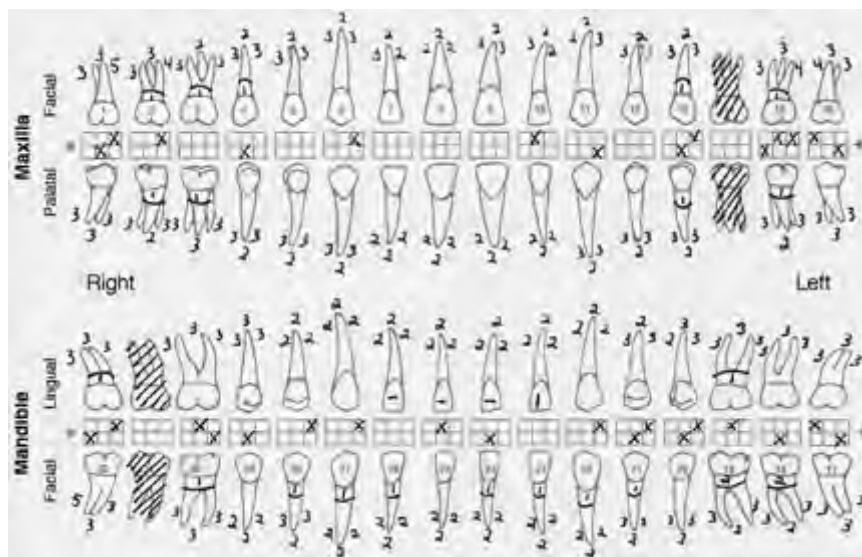
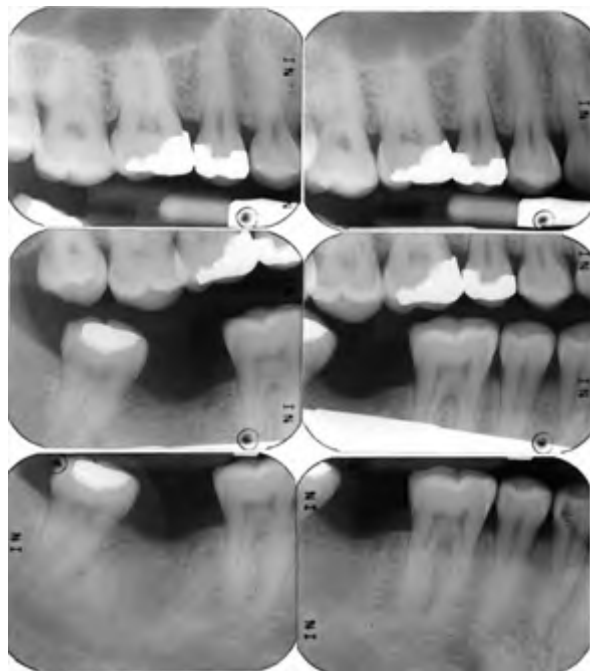
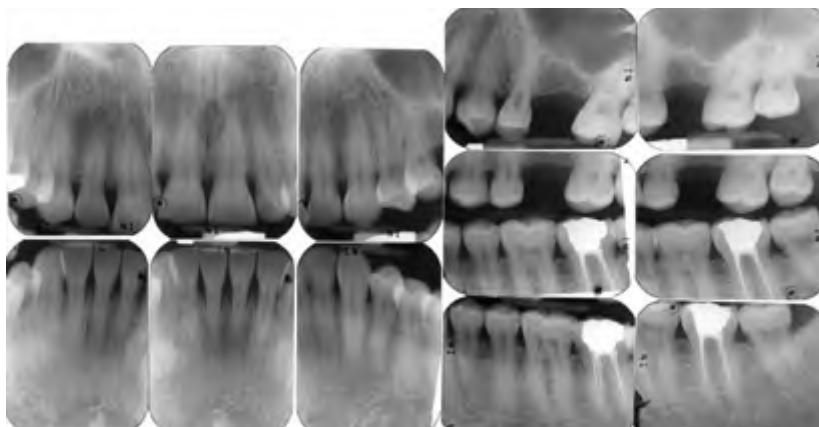
Medical History

- | | Yes | No |
|---|-------------------------------------|-------------------------------------|
| 1. Are you in good health? | ☒ | ☐ |
| 2. Has there been a change in your health the last year? | ☐ | ☒ |
| 3. Are you under the care of a physician? | ☒ | ☐ |
| 4. Have you been hospitalized in the past 10 years? | ☐ | ☒ |
| Reason for hospitalization: _____. | | |
| 5. Do you have or have you had any of the following conditions: | | |
| Damaged heart valves | ☒ | ☐ |
| Artificial heart valves | ☐ | ☒ |
| Heart murmur | ☒ | ☐ |
| Rheumatic heart disease | ☒ | ☐ |
| Heart attack | ☐ | ☒ |
| Angina | ☐ | ☒ |
| High blood pressure | ☒ | ☐ |
| Stroke | ☐ | ☒ |
| Asthma | ☐ | ☒ |
| Seizures | ☐ | ☒ |
| Fainting spells | ☐ | ☒ |
| Diabetes | ☐ | ☒ |
| Hepatitis/jaundice/liver disease | ☐ | ☒ |
| AIDS/HIV infection | ☐ | ☒ |
| Emphysema/bronchitis | ☐ | ☒ |
| Arthritis | ☐ | ☒ |
| Kidney disease | ☐ | ☒ |
| Tuberculosis | ☐ | ☒ |

- | | | |
|---|-------------------------------------|-------------------------------------|
| Low blood pressure | ☐ | ☒ |
| Sexually transmitted disease | ☐ | ☒ |
| Epilepsy | ☐ | ☒ |
| Cancer | ☐ | ☒ |
| Blood disorder/anemia | ☐ | ☒ |
| 6. Are you taking any of the following types of medications? | | |
| Antibiotics | ☐ | ☒ |
| Aspirin | ☐ | ☒ |
| Insulin | ☐ | ☒ |
| Nitroglycerin | ☐ | ☒ |
| Anticoagulants | ☐ | ☒ |
| Antihypertensives | ☒ | ☐ |
| Steroids | ☐ | ☒ |
| Oral contraceptives | ☐ | ☒ |
| Other, please name <u>Atenolol, Lasix (furosemide)</u> | | |
| 7. Do you have an allergy to any of the following? | | |
| Local anesthetics | ☐ | ☒ |
| Penicillin | ☒ | ☐ |
| Sulfa drugs | ☐ | ☒ |
| Barbiturates/sedatives | ☐ | ☒ |
| Aspirin | ☐ | ☒ |
| Narcotics/codeine | ☐ | ☒ |
| Rubber goods/latex | ☐ | ☒ |
| 8. Do you smoke or use any tobacco products? | ☐ | ☒ |
| 9. Are you pregnant? | ☐ | ☒ |
| 10. Do you wear any dental appliances? | ☐ | ☒ |
| 11. Have you had trouble associated with previous dental treatment? | ☐ | ☒ |
| If so, please describe _____. | | |

Dental History

Last dental visit 2 years ago.



1. The antibiotic regimen for this patient should include:
 - a. 2 g penicillin VK 1 hour prior to appointment
 - b. 2 g amoxicillin 1 hour prior to appointment
 - c. 600 mg clindamycin 1 hour prior to appointment
 - d. no prophylaxis necessary
2. All of the following predispose to bacterial endocarditis and require prophylaxis, except:
 - a. patent atrial septal defect
 - b. mitral valve prolapse with regurgitation
 - c. rheumatic fever
 - d. prosthetic heart valves
 - e. all the above
3. Atenolol and Lasix are (respectively):
 - a. calcium channel blocker and diuretic
 - b. calcium channel blocker and antihistamine
 - c. beta blocker and diuretic
 - d. alpha blocker and antihistamine
 - e. none of the above
4. To address the patient's chief complaint, the placement of an implant, abutment, and crown is discussed for the edentulous space at #14. What must be considered for that course of treatment?
 - a. caries on the distal of #13 and mesial of #14
 - b. the supereruption of #19
 - c. pneumatized left maxillary sinus
 - d. none of the above
5. After discussion, the patient would prefer to restore #31 with an FPD from #30 to #32. The dentist recommends porcelain fused to metal restoration. If the abutments are caries free, the reduction required for the prosthesis is:
 - a. 3 mm total reduction for 1 mm of metal and 2 mm porcelain
 - b. 2 mm total reduction for 0.5 mm of metal and 1.5 mm porcelain
 - c. 2 mm total reduction for 1 mm metal and 1 mm porcelain
 - d. 3 mm total reduction for 1.5 mm of metal and 1.5 mm of porcelain
 - e. none of the above
6. Porcelain obtains its strength from:
 - a. feldspar
 - b. glass
 - c. quartz
 - d. kaolin

7. Utilizing the diagnostic information on the distal bite wing, the next treatment for tooth #18 should include:
 - a. post, core, and crown
 - b. PFM crown
 - c. RCT retreatment
 - d. caries excavation and restoration
8. The radiopacity in the pulp chamber of tooth #3 is most likely a/an:
 - a. cementicle
 - b. denticle
 - c. odontogenic cyst
 - d. concretion
 - e. none of the above

1. **The answer is C.** Premed regimen:
 - amoxicillin 2.0 g 1 hour prior (kids 50 mg/kg)
 - clindamycin 600 mg 1 hour prior (kids 20 mg/kg)
 - azithromycin 500 mg 1 hour prior (kids 15 mg/kg)
 - clarithromycin 500 mg 1 hour prior (kids 15 mg/kg)All children's doses not to exceed adult dose.
2. **The answer is A.** Review the American Heart Association's guidelines for bacterial endocarditis prophylaxis.
3. Atenolol is a beta-1 receptor-specific blocker. Lasix (furosemide) is a loop diuretic. Both are used to treat hypertension.
4. **The answer is C.** Pneumatization of the sinus is of paramount importance when placing implant fixtures. At times, a sinus lift and bone grafts are indicated to provide adequate height and width for implant placement.
5. **The answer is B.** A PFM crown requires 0.5 mm metal and at least 1.5 mm of porcelain. If a preparation is underreduced, porcelain will most likely be overbulk to get the required thickness, resulting in an unaesthetic restoration.
6. **The answer is C.** Porcelain's strength comes from quartz. Feldspar lowers fusion temperature. Kaolin acts as a binder.
7. **The answer is D.** There's caries on the mesial of #18. It must be removed before any long-term restoration is placed. The caries is just in the enamel; thus, remineralization is also possible as a course of treatment.
8. **The answer is B.** Denticles are circular to oblong radiopacities that are radiographically visible in pulp chambers. Cementicles manifest on root surfaces. Concrecence is a fusion of the cemental surface of two teeth.

CASE 4

Patient Summary

Sex: F
Age: 35
Ht: 5'9"
Wt: 115 lb. (52 kg)

Vital Signs

BP: 116/74
Pulse: 68
Resp. Rate: 12

Chief Complaint

"I would like a checkup and have some work finished."

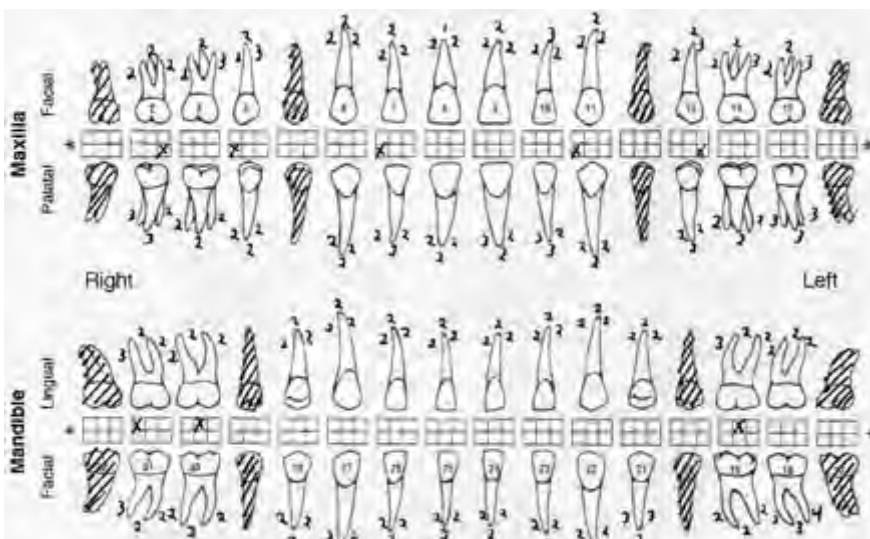
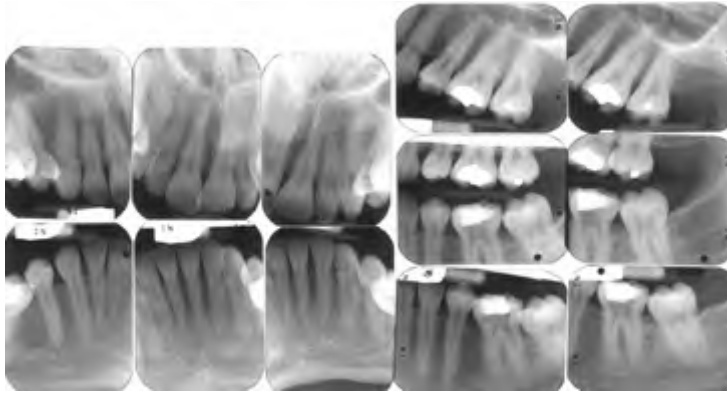
Medical History

	Yes	No
1. Are you in good health?	☒	☐
2. Has there been a change in your health the last year?	☐	☒
3. Are you under the care of a physician?	☐	☒
4. Have you been hospitalized in the past 10 years?	☐	☒
Reason for hospitalization: _____		
5. Do you have or have you had any of the following conditions:		
Damaged heart valves	☐	☒
Artificial heart valves	☐	☒
Heart murmur	☐	☒
Rheumatic heart disease	☐	☒
Heart attack	☐	☒
Angina	☐	☒
High blood pressure	☐	☒
Stroke	☐	☒
Asthma	☐	☒
Seizures	☐	☒
Fainting spells	☐	☒
Diabetes	☐	☒
Hepatitis/jaundice/liver disease	☐	☒
AIDS/HIV infection	☐	☒
Emphysema/bronchitis	☐	☒
Arthritis	☐	☒
Kidney disease	☐	☒
Tuberculosis	☐	☒
Low blood pressure	☐	☒

- | | | |
|--|-------------------------------------|-------------------------------------|
| Sexually transmitted disease | ☐ | ☒ |
| Epilepsy | ☐ | ☒ |
| Cancer | ☐ | ☒ |
| Blood disorder/anemia | ☐ | ☒ |
| 6. Are you taking any of the following types of medications? | | |
| Antibiotics | ☐ | ☒ |
| Aspirin | ☐ | ☒ |
| Insulin | ☐ | ☒ |
| Nitroglycerin | ☐ | ☒ |
| Anticoagulants | ☐ | ☒ |
| Antihypertensives | ☐ | ☒ |
| Steroids | ☐ | ☒ |
| Oral contraceptives | ☐ | ☒ |
| Other, please name _____ | | |
| 7. Do you have an allergy to any of the following: | | |
| Local anesthetics | ☐ | ☒ |
| Penicillin | ☐ | ☒ |
| Sulfa drugs | ☐ | ☒ |
| Barbiturates/sedatives | ☐ | ☒ |
| Aspirin | ☐ | ☒ |
| Narcotics/codeine | ☐ | ☒ |
| Rubber goods/latex | ☐ | ☒ |
| 8. Do you smoke or use any tobacco products? | ☐ | ☒ |
| 9. Are you pregnant? | ☐ | ☒ |
| 10. Do you wear any dental appliances? | ☐ | ☒ |
| 11. Have you had trouble associated with previous dental treatment? | ☒ | ☐ |
| If so, please describe: <u>My last dentist was rude and she hurt me.</u> | | |

Dental History

Last dental visit about 1 year ago.



1. The dentist asks the patient, "Would you please tell me more about your experience with your last dentist?" This type of question is:
 - a. a leading question
 - b. a laundry list question
 - c. a probing question
 - d. a direct question
2. The patient complains of pain when she drinks cold liquids in the region of tooth #31. The dentist performs a cold test on #31 and elicits a 24-second response similar to her reported symptom. The most likely diagnosis is:
 - a. irreversible pulpitis
 - b. reversible pulpitis
 - c. pulpal necrosis
 - d. none of the above
3. During the course of the clinical exam, the dentist observes a proximal carious lesion on the mesial of #22. The patient elects for a composite restoration. The enamel of the composite preparation is etched with _____ and upon washing and drying, should appear _____.
 - a. acetic acid; yellow and shiny
 - b. phosphoric acid; yellow and shiny
 - c. phosphoric acid; white and chalky
 - d. acetic acid; white and chalky
 - e. none of the above
4. The dentist continues treatment on tooth #19 with a pulpectomy. The most common cause of failure in root canal therapy is:
 - a. filler leakage
 - b. undiagnosed fracture
 - c. inadequate disinfection of the canal
 - d. none of the above
5. The absence of maxillary first premolars and mandibular second premolars is suggestive of:
 - a. rampant caries that lead to extractions
 - b. xerostomia and subsequent extractions
 - c. loss of teeth due to trauma
 - d. serial extractions for orthodontic treatment
6. The radiolucency in the pulp chamber of tooth #2 may be a/an:
 - a. pulp stone
 - b. denticle
 - c. cementicle
 - d. a or b
 - e. a or c

7. Serial extractions may involve all the following, except:
 - a. extraction of primary molars
 - b. extraction of primary canines
 - c. extraction of second premolars
 - d. extraction of first premolars
 - e. extraction of first premolars after permanent canines erupt
 - f. extraction of first premolars before permanent canines erupt
8. Recurrent caries was detected under the amalgam restoration on tooth #15. During caries removal, the dentist made a pinpoint exposure of the pulp. Which of the following is false with regard to the success of a direct pulp capping procedure?
 - a. The tooth should be asymptomatic before permanent restoration is placed.
 - b. An iatrogenic exposure has a worse prognosis than a carious one.
 - c. Direct pulp caps have been shown to be more successful in permanent teeth than primary teeth.
 - d. All the above are false.
 - e. All the above are true.
9. What is the most significant error in the maxillary left canine radiographic view?
 - a. overlap of the lateral incisor and canine
 - b. poor centering of the film
 - c. the film was placed backward in the mouth
 - d. incorrect dot placement
10. What is the maximum dose of anesthetic that can be administered to this patient (2% lidocaine 1:100,000 epinephrine, 1.8 mL/carpule)?
 - a. 4.5 mg/kg
 - b. 5.5 mg/kg
 - c. 6.0 mg/kg
 - d. 6.5 mg/kg
 - e. 7.0 mg/kg

1. **The answer is C.** A leading question has suggestive information in it. (i.e., You don't brush your teeth very often, do you?) A laundry list question is like a multiple-choice question. (i.e., Does it hurt when you chew, eat or drink?) Direct questions look for a specific piece of information. (i.e., Does it hurt when I probe here?)
2. **The answer is A.** Irreversible pulpitis elicits a response to the cold test lasting longer than 10 seconds that is greater than control teeth. Reversible pulpitis is usually less than 10 seconds but similar to controls. Necrotic tooth has a negative response to the cold test.
3. **The answer is C.** Phosphoric acid is used to etch enamel and the appearance after washing and drying is white and chalky (a frosty appearance).
4. **The answer is C.** Inadequate disinfection of the canals is the most common cause of failure for RCT. Obturator leakage is the second most common.
5. **The answer is D.** There is no evidence to suggest any of the first three choices. Serial extractions are usually in the following sequence:
 1. primary cuspid
 2. primary first molar
 3. first bicuspid
6. **The answer is D.** A radiographic calcification in the pulp chamber may be a true denticle or calcification of pulp tissue (pulp stone).
7. **The answer is E.** Extraction of first premolars is necessary to provide space for incoming permanent canines. This should be done before these canines erupt.
8. **The answer is B.** A carious exposure of the pulp generally has a worse prognosis than an iatrogenic exposure because bacteria have already progressed deep into the dentin. An iatrogenic exposure is likely more hygienic.
9. **The answer is A.** The periapical maxillary lateral view is imperative to evaluate the contact area of the cuspid and lateral incisor. Caries undetected via transillumination may go undetected if overlap is present.
10. **The answer is E.** The maximum amount of this anesthetic for a normal adult is 7 mg/kg. For children, it is about 4 mg/kg.

CASE 5

Patient Summary

Sex: F
Age: 21
Ht: 5'5"
Wt: 115 lb. (52 kg)

Vital Signs

BP: 108/74
Pulse: 60
Resp. Rate: 10

Chief Complaint

"I would like to replace my missing teeth."

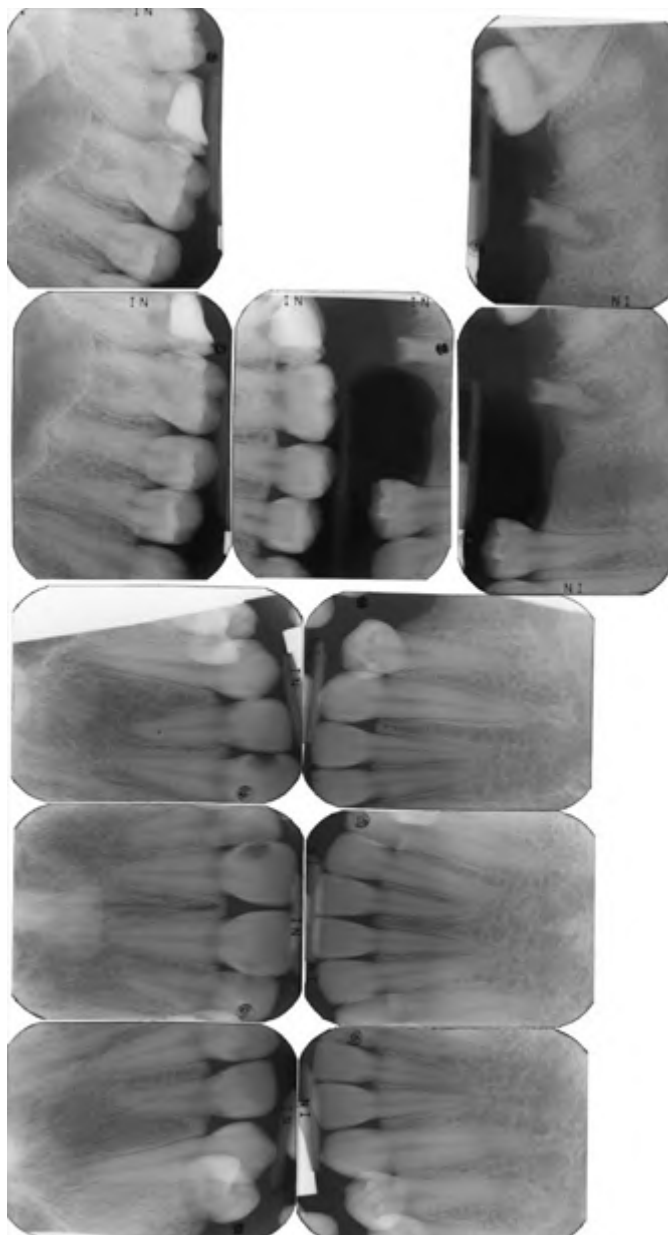
Medical History

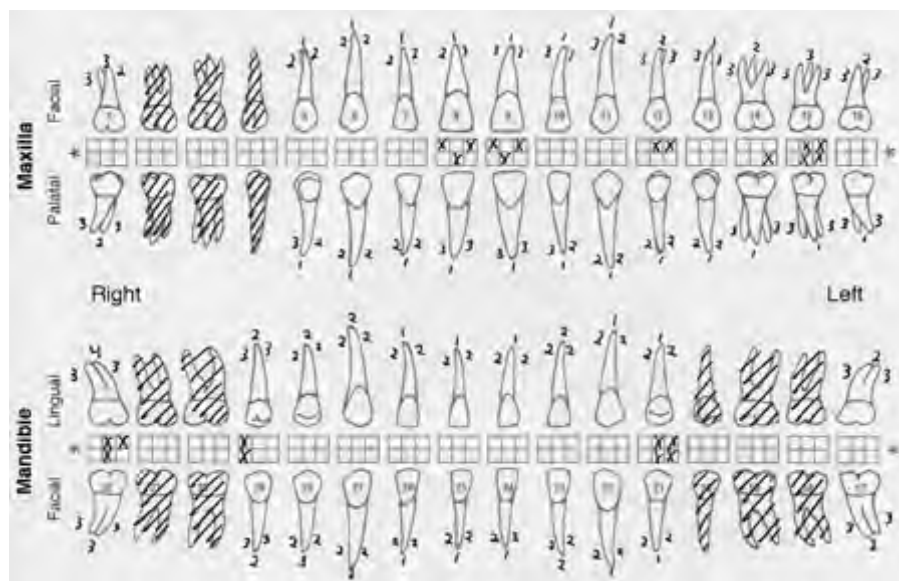
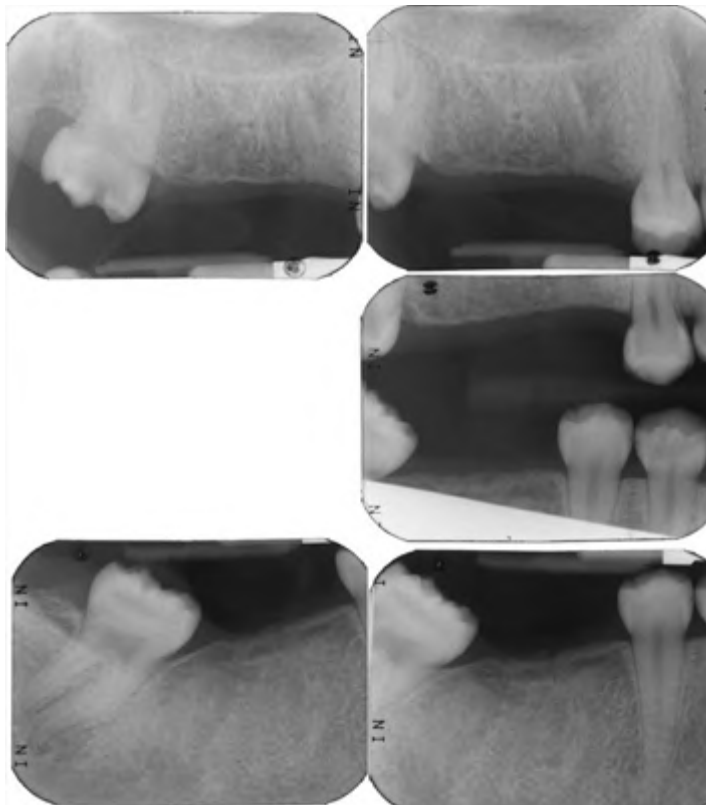
- | | Yes | No |
|---|-------------------------------------|-------------------------------------|
| 1. Are you in good health? | ☒ | ☐ |
| 2. Has there been a change in your health the last year? | ☐ | ☒ |
| 3. Are you under the care of a physician? | ☒ | ☐ |
| 4. Have you been hospitalized in the past 10 years? | ☐ | ☒ |
| Reason for hospitalization: _____. | | |
| 5. Do you have or have you had any of the following conditions: | | |
| Damaged heart valves | ☐ | ☒ |
| Artificial heart valves | ☐ | ☒ |
| Heart murmur | ☒ | ☐ |
| Rheumatic heart disease | ☐ | ☒ |
| Heart attack | ☐ | ☒ |
| Angina | ☐ | ☒ |
| High blood pressure | ☐ | ☒ |
| Stroke | ☐ | ☒ |
| Asthma | ☐ | ☒ |
| Seizures | ☐ | ☒ |
| Fainting spells | ☐ | ☒ |
| Diabetes | ☐ | ☒ |
| Hepatitis/jaundice/liver disease | ☐ | ☒ |
| AIDS/HIV infection | ☐ | ☒ |
| Emphysema/bronchitis | ☐ | ☒ |
| Arthritis | ☐ | ☒ |

Kidney disease	☐	☒
Tuberculosis	☐	☒
Low blood pressure	☐	☒
Sexually transmitted disease	☐	☒
Epilepsy	☐	☒
Cancer	☐	☒
Blood disorder/anemia	☐	☒
6. Are you taking any of the following types of medications?		
Antibiotics	☐	☒
Aspirin	☐	☒
Insulin	☐	☒
Nitroglycerin	☐	☒
Anticoagulants	☐	☒
Antihypertensives	☐	☒
Steroids	☐	☒
Oral contraceptives	☒	☐
Other, please name _____.		
7. Do you have an allergy to any of the following:		
Local anesthetics	☐	☒
Penicillin	☐	☒
Sulfa drugs	☐	☒
Barbiturates/sedatives	☐	☒
Aspirin	☐	☒
Narcotics/codeine	☐	☒
Rubber goods/latex	☐	☒
8. Do you smoke or use any tobacco products?	☒	☐
9. Are you pregnant?	☐	☒
10. Do you wear any dental appliances?	☐	☒
11. Have you had trouble associated with previous dental treatment?	☐	☒
If so, please describe _____.		

Dental History

Last dental visit 2 years ago.





QUESTIONS

1. Upon questioning, the patient informs the dentist she is always thirsty, and often sucks on hard candy. During the appointment, she excuses herself to use the restroom several times. This information should alert the dentist to possible:
 - a. lupus
 - b. nephritis
 - c. jaundice
 - d. diabetes
 - e. none of the above
2. The patient recently discovered she has mitral valve prolapse with regurgitation. The dentist prescribes amoxicillin for subsequent visits. The patient should be informed:
 - a. she should use secondary methods of contraceptives when taking amoxicillin
 - b. to take the medication 1 hour before and immediately after her appointment
 - c. there is a high likelihood she will experience thrush
 - d. none of the above
 - e. all the above
3. With regard to the missing dentition, the first course of action the dentist should take is:
 - a. ask the patient how she would like to restore them
 - b. take preliminary impressions and fabricate diagnostic casts
 - c. ask the patient to demonstrate brushing and flossing technique
 - d. ascertain how the teeth were lost
 - e. examine the area for restorative space considerations
4. The dentist refers the patient to an oral surgeon for extraction of root tips at #18, #19 and tooth #17 for prosthetic reasons. The oral surgeon refers to these radiographs. The oral surgeon should:
 - a. place the patient on antibiotics to limit the periapical infection first
 - b. proceed with the simple extraction procedure
 - c. proceed with the soft tissue-impacted extraction
 - d. have a new radiograph taken
 - e. none of the above
5. The dentist examines the periapical radiolucency at root tip in area of #19. The patient reports no symptoms. The differential diagnosis should not include:
 - a. periapical cyst
 - b. periapical scar
 - c. periapical abscess
 - d. periapical granuloma
 - e. none of the above (All should be included.)

6. What is the most likely reason the patient experiences no symptoms associated with this root tip?
 - a. The infection has run its course and is now a scar.
 - b. The infection is draining itself through the cavity.
 - c. The root tip is necrotic.
 - d. none of the above
7. The dentist would like to restore the dentition at the area of #18, #19, and #20 with a removable partial denture. Which of the below may become the MOST problematic for this restoration?
 - a. the extrusion of #13, #14, and #15
 - b. the retained root tips at #18 and #19
 - c. the periapical radiolucency in the area
 - d. the pulpotomy of #15
 - e. the pneumatized left maxillary sinus
8. If the deep class III carious lesions in tooth #9 were cultured, which of the following bacteria is most likely to be cultured in the most abundance?
 - a. streptococcus mutans
 - b. lactobacillus *paracasei*
 - c. porphyromonas gingivalis
 - d. treponema denticola
 - e. all are equally likely to be cultured in the most abundance
9. The patient enquires about implant restorations. In which areas are implants least successfully osseointegrated?
 - a. maxillary anterior
 - b. mandibular anterior
 - c. maxillary posterior
 - d. mandibular posterior
10. During the interview, the dentist enquires further about the patient's smoking habit. The patient responds that she has smoked for 2.5 years, now smokes 1 pack a week, and smokes only when she drinks alcohol. The dentist informs the patient of the cancer risks associated with this behavior. Which of the following statements is true?
 - a. Oral cancer most frequently occurs on the floor of the mouth.
 - b. Oral cancer occurs least frequently on the lateral borders of the tongue.
 - c. Oral cancer occurs least frequently on the floor of the mouth.
 - d. Alcohol use and smoking tobacco exacerbate each other's carcinogenic effect.
 - e. There is no link between tobacco smoking and alcohol use in carcinogenicity.

1. **The answer is D.** The “three poly’s” are ear marks for diabetes: polydipsia (abnormally large fluid intake), polyurea (excessive urination), and polyphagia (excessive hunger).
2. **The answer is A.** The patient reported use of oral contraceptives. Amoxicillin may interact with and affect the efficacy of oral contraceptives. The likelihood of thrush is low.
3. **The answer is D.** This patient is very young for her oral/dental condition. In any case, before attempting to replace caries and absent dentition, an attempt should be made to discover how or why the problems are present. The etiological agents responsible for the condition must be addressed in order to treat the disease, and not just the symptoms.
4. **The answer is D.** The radiograph of tooth #17 is missing the apices and is therefore undiagnostic. A complete view via another periapical radiograph or a panoramic radiograph is therefore necessary before anything further.
5. **The answer is E.** All are included in the differential. A biopsy would be necessary to make a true diagnosis.
6. **The answer is B.** The site is one of active infection and although the tooth is necrotic, that is not the reason for lack of symptoms. The infection is draining upward into the oral cavity through the retained root.
7. **The answer is A.** Extruded opposing dentition distorts the curve of Spee and may necessitate enameloplasty, RCT, or extraction to accommodate new space requirements for restorative dentition.
8. **The answer is B.** Lactobacillus species reside in deep carious lesions. *Strep. Mutans* play a role in the beginning of the caries process. *P. Gingivalis* and *T. Denticola* play roles in periodontal disease.
9. **The answer is C.** Implant are most successful in the mandibular anterior and least successful in the maxillary posterior.
10. **The answer is D.** Oral cancer most frequently occurs on the lateral borders of the tongue and second most frequently on the floor of the mouth. Studies have shown that alcohol use with tobacco use increases the chances of developing such a lesion.

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